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A.E.S.

TRANSACTIONS

OF THE

AMERICAN PEDIATRIC SOCIETY

THIRTEENTH SESSION

HELD AT NIAGARA FALLS, N.Y., MAY 27, 28 AND 29, 1901

EDITED BY

WALTER LESTER CARR, M.D.

VOLUME XIII.

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PRESIDENTS.

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1890 J. Lewis Smith, M.D.
1891. T. M. Rotch, M.D.
1892. Wm. Osler, M.D.
1893. A. D. Blackader, M.D.
1894. John M. Keating, M.D.
1895. F. Forchheimer, M.D.
1896. Joseph O'Dwyer, M.D.
1897. Samuel S. Adams, M.D.
1898. L. Emmett Holt, M.D.
1899. Wm. P. Northrup, M.D.
1900. Henry Koplik, M.D.
1901. Wm. D. Booker, M.D.
1902. W. S. Christopher, M.D.

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Wm. Osler, M.D.
C. P. Putnam, M.D.
F. Forchheimer, M.D.
J. C. Wilson, M.D.
F. M. Crandall, M.D.

MEETING PLACES.

1888. Washington, D. C. (Organization), September 18.
1889. Washington and Baltimore, September 20 and 21.
1890. New York, June 3 and 4.
1891. Washington, September 22 and 25.
1892. Boston, May 2, 3, and 4.
1893. West Point, N. Y., May 24, 25 and 26.
1894. Washington, May 29 and June 1.
1895. Virginia Hot Springs, May 27, 28 and 29.
1896. Montreal, May 25, 26 and 27.
1897. Washington, May 4, 5 and 6.
1898. Cincinnati, June 1, 2 and 3.
1899. Deer Park, June 27, 28 and 29.
1900. Washington, May 1, 2 and 3.
1901. Niagara Falls, May 27, 28 and 29.
1902. Boston, May 26, 27 and 28.

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Delegate to the Congress of American Physicians and Surgeons

A. JACOBI, M.D.

Alternate, - - - - - J. P. CROZER GRIFFITH, M.D.

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PACKARD, FREDERICK A., M.D.....258 South Eighteenth Street, Philadelphia
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WILLIAMS, HAROLD, M.D.....528 Beacon Street, Boston
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WINTERS, J. E., M.D.....25 West Thirty-seventh Street, New York
YALE, LEROY M., M.D... .....432 Madison Avenue, New York

Deceased.

JOHN A. JEFFRIES, M.D.
Born, September 2, 1859,
Died, March 26, 1892.

THOMAS F. SHERMAN, M.D.
Born, March 17, 1856,
Died, September 26, 1893.

JOHN M. KEATING, M.D.
Born, April 20, 1852,
Died, November 17, 1893.

CHARLES WARRINGTON EARLE, M.D.
Born, 1845,
Died, November 19, 1893.

J. LEWIS SMITH, M.D.
Born, October 15, 1827,
Died, June 9, 1897.

JOSEPH O'DWYER, M.D.
Born, October 12, 1841,
Died January 7, 1898.

JOHN HENRY FRUITNIGHT, M.D.
Born, November 9, 1851,
Died, December 18, 1900.

MINUTES OF THE THIRTEENTH ANNUAL MEETING OF THE AMERICAN PEDIATRIC SOCIETY.

Held at Niagara Falls, N. Y., May 27, 28 and 29, 1901.

The meeting was called to order by the President, Dr. Wm. D. Booker, of Baltimore. The minutes of the twelfth annual meeting were approved as published in ARCHIVES OF PEDIATRICS.

The following members were present: G. N. Acker, M.D., Washington; S. S. Adams, M.D., Washington; A. Baines, M. D., Toronto; W. D. Booker, M.D., Baltimore; E. M. Buckingham, M.D., Boston; W. L. Carr, M.D., New York; H. D. Chapin, M.D., New York; W. S. Christopher, M.D., Chicago; F. S. Churchill, M.D., Chicago; A. C. Cotton, M.D., Chicago; J. Dorning, M.D., New York; R. G. Freeman, M.D., New York; E. E. Graham, M.D., Philadelphia; J. P. C. Griffith, M.D., Philadelphia; S. McC. Hamill, M.D., Philadelphia; L. E. Holt, M.D., New York; C. G. Kerley, M.D., New York; H. Koplik, M.D., New York; J. L. Morse, M.D., Boston; W. P. Northrup, M.D., New York; F. A. Packard, M.D., Philadelphia; C. P. Putnam, M.D., Boston; B. K. Rachford, M.D., Cincinnati; T. M. Rotch, M.D., Boston; E. W. Saunders, M.D., St. Louis; I. M. Snow, M.D., Buffalo; C. W. Townsend, M.D., Boston; A. H. Wentworth, M.D., Boston; J. P. West, M.D., Bellaire; H. Williams, M.D., Boston; J. C. Wilson, M.D., Philadelphia; J. E. Winters, M.D., New York.

Upon motion by Dr. Snow, members of the profession from Canada and the State of New York were invited to attend the meetings and take part in the discussion.

FIRST SESSION.—MAY 27.

The annual address of the President, entitled "The Early History of the Summer Diarrheas of Infants," was read by Dr. Wm. D. Booker, of Baltimore.

Dr. Allen Baines, of Toronto, reported a case of "General Arteriosclerosis in a Boy Aged Ten Years," and showed microscopic specimens.

Dr. T. M. Rotch, of Boston, read a paper entitled "Pernicious Anemia in Infancy (Preliminary Report of a Case)," by himself and Dr. M. Ladd.

Discussion by Drs. Wentworth, Koplik, Morse, Northrup, Churchill, Kerley, Townsend, Winters and Rotch.

Dr. F. S. Churchill, of Chicago, read a paper entitled "So-called Cyclical Albuminuria with Preliminary Report of a Case."

Discussion by Drs. Dorning, Packard, Winters, Koplik and Churchill.

Dr. R. G. Freeman read a paper entitled "An Account of a Mild Epidemic Due to Some Unrecognized Infection."

Discussion by Drs. Packard, Kerley, Buckingham, Wentworth, Adams and Freeman.

SECOND SESSION.—MAY 27.

Dr. J. P. C. Griffith, of Philadelphia, read papers entitled (*a*) "Report of a Case of Appendicitis in a Child of Three Months." (*b*) "Report of a Case of Long Continued Laryngeal Stenosis of Uncertain Nature."

Discussion of the first paper by Dr. Shaw (guest).

Discussion of the second paper by Drs. Rachford, Rotch, Buckingham, Koplik and Griffith.

Dr. A. C. Cotton, of Chicago, read a paper entitled "Amaurotic Family Idiocy" and gave a report on "A Monster" with exhibition of specimen and photographs.

Discussion by Dr. Morse.

Dr. J. Park West, of Bellaire, read a paper entitled "A Note on the Little Finger of the Mongolian Imbecile and Normal Children" and showed photographs.

Discussion by Drs. Rotch and Koplik.

Dr. B. K. Rachford, of Cincinnati, read two papers entitled (*a*) "Maternal Impressions, Report of Cases," and (*b*) "Hare-Lip."

Discussion of the first paper by Drs. Rotch, Wentworth and Rachford.

THIRD SESSION.—MAY 27.

Drs. F. A. Packard and Alfred Hand, Jr., of Philadelphia, presented a paper on "The Pathological Anatomy of Cretinism," which was read by Dr. Packard.

Drs. Henry Koplik and I. Lichtenstein, of New York, presented a paper, entitled "A Contribution to the Symptomatology of Cretinism," which was read by Dr. Koplik.

Discussion of both papers by Drs. Cotton, Northrup, Christopher, West, Booker, Freeman, Adams, Packard and Koplik.

Dr. W. P. Northrup, of New York, read a paper entitled "Glass Sun Rooms on City Roofs, or Winter Play Houses," with illustrations.

Discussion by Drs. Putnam, Adams, Winters, Rotch, Griffith and Northrup.

FIRST SESSION.—MAY 28.

Dr. Charles G. Kerley, of New York, read a paper entitled "A Study of 555 Cases of Summer Diarrhea."

Discussion by Drs. Griffith, Koplik, Buckingham, Holt, Northrup, Chapin, Freeman, Winters, Adams, Cotton, Morse, Dorning, Rotch, Wentworth, Saunders, Williams, Townsend and Kerley.

Dr. C. W. Townsend, of Boston, read a paper entitled "The Feeding of an Incubator Baby."

Dr. H. D. Chapin, of New York, read a paper on "The Place of Cereals in Infant Feeding."

Discussion on both papers was postponed until the afternoon session.

SECOND SESSION.—MAY 28.

The Vice-President, Dr. Packard, occupied the chair.

Discussion of Dr. Townsend's paper by Drs. Chapin, Morse, Rotch and Townsend.

Discussion of Dr. Chapin's paper by Drs. Rotch, Koplik, Griffith, Holt, Winters, Saunders and Chapin.

Dr. E. W. Saunders, of St. Louis, showed "A Specimen of Congenital Hypertrophic Pyloric Stenosis," and gave a report of the case.

Discussion by Drs. Holt and Saunders.

The following papers were read by title:

Dr. D. J. M. Miller, of Philadelphia, "A Case of Hemorrhagic Nephritis Complicating Influenza in a Thirteen Months' Old Baby."

Dr. Wm. Osler, of Baltimore, "Congenital Absence of the Abdominal Muscles with Distended and Hypertrophied Urinary Bladder in a Child of Six Years."

Dr. A. Jacobi, of New York, "Milk Sugar."

Dr. A. Seibert, of New York, "The Treatment of the Nasopharynx in Scarlatina."

Dr. F. Huber, of New York, "Etiology of Rectal Polypi in Children."

Dr. Wm. Osler, of Baltimore, "The Visceral Lesions of the Erythema Group of Skin Diseases in Young Children."

Dr. F. Forchheimer, of Cincinnati, "The Use of the Term Enanthem."

Dr. John L. Morse, of Boston, "An Analysis of 32 Cases of Congenital Heart Disease."

Dr. F. Huber, of New York, (*a*) "Photographs of a Case of Infantile Scurvy and Marasmus." (*b*) "History of a Case of Gangrene of the Lung."

Dr. Walter Lester Carr, of New York, "A Case of Pulmonary Gangrene in a Baby."

Dr. George N. Acker, of Washington, "Multiple Arthritis in a Child Two Years Old Suffering from Gonorrheal Vaginitis."

Dr. I. M. Snow, of Buffalo, "Bulbar Symptoms in the Newly-Born."

Dr. B. K. Rachford, of Cincinnati, "The Treatment of Tuberculosis."

Dr. C. P. Putnam, of Boston, "A Case of Pneumococcus Infection."

FIRST SESSION.—MAY 29.

Dr. J. Lovett Morse, of Boston, read a report of "A Case of Arsenical Poisoning in an Infant of Six Months."

Dr. S. S. Adams, of Washington, read a paper entitled "Great Fluctuations in Temperature in the Terminal Stage of Pulmonary Tuberculosis."

Discussion by Drs. Griffith, Wentworth, Adams and Holt.

Dr. L. E. Holt, of New York, read two papers entitled (*a*) "Note on the Treatment of Hemorrhage in the New-Born by

Suprarenal Extract," and (*b*) "Prolonged Temperature from an Unusual Cause."

Discussion of the first paper by Drs. Morse, Rotch, Carr, Wilson, Chapin and Holt.

Discussion of the second paper by Drs. Morse, Chapin and Wilson.

Dr. Harold Wilson, of Boston, read a paper entitled "Measles Complicated by Appendicitis."

Dr. W. S. Christopher, of Chicago, read a paper entitled "Physical Measurements in Puberty, their Significance, Variation and Applications."

Discussion by Drs. Holt and Booker.

EXECUTIVE SESSION.—MAY 29.

The report of the Council was presented by its Chairman, Dr. Rachford. Upon motion of Dr. Adams, it was adopted as read. On nomination of the Council the following officers were elected for the ensuing year:

President, - - W. S. CHRISTOPHER, M.D., Chicago.
First Vice-President, C. W. TOWNSEND, M.D., Boston.
Second Vice-President, JOHN DORNING, M.D., New York.
Secretary, - - SAMUEL S. ADAMS, M.D., Washington.
Treasurer, - - J. PARK WEST, M.D., Bellaire, Ohio.
Recorder and Editor, WALTER LESTER CARR, M.D., New York.
Member of the Council, T. M. ROTCH, M.D., Boston.

Delegate to Congress of American Physicians and Surgeons, A. Jacobi, M.D.; Alternate, J. P. C. Griffith, M.D.

Member of Committee of Arrangements, S. S. Adams, M.D.

Elected to Membership, David L. Edsall, M.D., of Philadelphia.

Place and Time of Meeting, *Boston,* May 26, 27, 28, 1902.

Assessment for Ensuing Year—*Five Dollars.*

The offer of ARCHIVES OF PEDIATRICS to print the Transactions as in previous years, was accepted.

The President appointed Drs. Griffith, Holt and Freeman a

committee for the purpose of indexing the volumes of the Transactions.

Members of the Society having extra volumes of the Transactions were requested to notify the Secretary.

The following resolutions on the death of Dr. Fruitnight were presented by Dr. Adams and adopted:

RESOLUTIONS.

WHEREAS this Society learns with deep regret of the death of Dr. J. Henry Fruitnight, of New York, one of the founders of this body, therefore,

Be it Resolved, That this Society expresses its loss in the death of such a useful member and earnest worker for the good of children; and it is hereby

Resolved, That we extend our sincere sympathy to the relatives of Dr. Fruitnight, that these resolutions be spread upon the minutes and published in the Transactions, and that a copy be sent to the family of the deceased.

WALTER LESTER CARR, M.D.,

Recorder.

JOHN HENRY FRUITNIGHT, M.D.

BORN, NOVEMBER 9, 1851—DIED, DECEMBER 18, 1900.

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THE EARLY HISTORY OF THE SUMMER DIARRHŒAS OF INFANTS.

BY W. D. BOOKER, M.D ,

Clinical Professor of Pediatrics, Johns Hopkins University, Baltimore, Md.

GENTLEMEN: I appreciate the honor you have conferred upon me in electing me to the highest office in your gift, and for this evidence of your esteem and confidence, I thank you.

Since our last meeting, the Society has sustained a severe loss in the death of Dr. J. H. Fruitnight, a conscientious and industrious worker, an active and faithful member of this Society, and a regular contributor to the success of its meetings.

For many years I have been deeply interested in the summer diarrhœas of infants, and since my attention was first directed to this subject, I have been impressed with the obscurity of the early history of these affections, and the difficulty of tracing the development of our knowledge concerning them. The literature on this subject can be found only in old books and journals, which are scattered in different libraries throughout this country, and are becoming scarcer and more difficult to obtain every year.

In selecting a subject for my address on this occasion, it occurred to me that a brief review of this literature might not be unworthy of your attention.

About the middle of the Eighteenth Century there occurred, in the towns and cities along the Atlantic coast of the United States of America, a serious disease among infants which had not been observed before. It was characterized more especially by vomiting and diarrhœa, and was confined almost exclusively to children in the first two years of life. Its incidence was limited to the summer months, and it appeared each year in an epidemic form with such regularity that it was looked for as an annual visitor—"the dread of parents and the opprobrium of physicians." It was thought to be peculiar to America, "and was viewed in the light of one of those physical occurrences which occasionally give rise to diseases before unknown, while others from causes equally unscrutable, disappear." This opinion, although an

erroneous one, prevailed for many years, and has rendered it a difficult task to trace the history of the malady to its origin. The disease was not recognized in Europe; it was unknown to the aborigines of our continent; and the first settlers of the colonies have left us no record of its existence. It was not until the people began to collect in towns and villages that the disease was engendered, and "it had no local habitation or name" until it was described by Benjamin Rush in 1777 in a paper entitled "An Inquiry into the Cause and Cure of The Cholera Infantum."* In Philadelphia it was called "vomiting and purging of children," and also from the regularity of its appearance in the summer months, "the disease of the season." In Charleston, S. C., it was distinguished as the "April and May disorder" since it was more especially prevalent in these two months.

It seems strange that a disease so familiar to us as cholera infantum, so striking in its symptoms, and frightful in its mortality, should have escaped professional attention for so long a time; nor is it a less remarkable fact that after its importance had been made known by American physicians, the disorder should have remained almost entirely unappreciated in Europe for half a century. If we seek an explanation for this strange neglect, it may be found in the low estimate formerly placed upon the lives of infants, and the consequent indifference to their diseases in general, and especially to those of the digestive tract, which were relegated for the most part to the care of mothers and ignorant nurses.

That the summer diarrhœas of children received an earlier recognition in America may be attributed to the importance to a new country of increase of population, as well as to the greater prevalence and more serious nature of the disease. It would almost seem as if some strange fate had destined that throughout its history, at all times, the medical profession should be too blind or too indifferent to recognize its vast importance as a scourge to the human race. Even to-day when the pathologist and bacteriologist are revolutionizing our knowledge on so many subjects in medicine, the summer diarrhœas of infancy which is so appropriately within their sphere of work, have not received

* "Cholera infantum" was used by Dr. Rush and his cotemporaries as a generic term to embrace the group of affections which, at the present time, are classed under the head of summer diarrhœas of infants. The term is used in this article in the sense intended by Dr. Rush.

any serious consideration at their hands. Whatever has been learned of their nature and etiology through the application of modern facilities for investigation, we owe entirely to the work of the clinician. While we have no clear history of the disease previous to the time it was recognized in this country, references found in the literature to the prevalence of diarrhœal troubles in infants in the summer months indicate its existence in all ages and in almost all countries.

Aretaeus the Cappadocian, who wrote in the Second Century of our era, says in his chapter on cœliac affections: "Children are subject to continual diarrhœa from an ephemeral intemperance of food; but in their case the disease is not seated in the cavity of the stomach. Summer engenders the disease more than any other season, autumn next; and the coldest season, winter, also, if the heat be almost extinguished."

Harris in his "De Morbis Acutis Infantum," published in London in 1650 says: "From the middle of the month of July to the middle of September, gripes of the belly are so mortal and epidemical to children, (their strength being quite exhausted by the heat) that more of them die yearly in this one month than any three or four months of the year."

Cleghorn writing on the "Epidemical Diseases of Minorca," from 1744 to 1749 inclusive, describes an epidemic form of diarrhœa in that island which occurs in children in the summer months, and appears to be identical with cholera infantum. He says: "As the summer and autumnal weather of one year never varies from that of another, so the same tribe of distempers return regularly with the season. Towards the end of June the young children (who suffer first by excessive heat or cold), are attacked with a vomiting and purging, and periodical fever, often of the erratic kind and without any fixed type. In 1747 the extraordinary heat of May ushered in the summer diseases sooner than usual, for about the end of that month the cholera morbus carried off many children, and in June the tertian began. In 1748 the summer proved very unhealthy to children, many of them dying of cholera morbus and periodical fever. In 1749 the drought was so great that the harvest failed. At the same time it was excessively hot in June, and the weather had seldom been felt so excessively hot and sultry as in this year in July. The medium height of the temperature was, in July, about one degree and a half greater than it had been in the six previous summers.

But notwithstanding this excessive heat in June and July, there were some specimens of the summer diseases, but so few that they scarcely deserve to be called epidemical; while in the preceding summers, one of which was not as hot as usual, the cholera was very fatal."

Rosenstein devotes forty pages of his book on diseases of children, published in Stockholm in 1750, to diarrhœal affections, of which fourteen different kinds are described. These are distinguished from an etiological standpoint. The fifth kind, diarrhœa biliosa, he says, comes from overloading the stomach, especially with fatty food; also from other causes, as the effect of a hot and dry summer, etc. Special caution is given to relieve a protracted diarrhœa in July and August, when it is hot in the day and cool in the evening, as it may easily change to the dysentery.

The writings of American physicians on cholera infantum were noticed in Germany at an earlier period than in other European countries, although they failed to arouse any enthusiasm on the subject, and it was many years before the importance of the disease was recognized. They succeeded, however, in calling attention to the subject, which resulted in positive evidence of the existence of this disease in Germany at the time it was first observed in America.

Girtanner's book on the diseases of children, published in Berlin, in 1792, contains a full abstract of the original article on cholera infantum by Rush, and the author says that, while the disease is of frequent occurrence in America, it is but seldom seen in Germany. Reusch describes an epidemic form of diarrhœa which occurred in children, from six months to two years of age, in Königsburg in the summer of 1812. Henke's *Handbuch der Kinderkrankheiten*, published in Berlin, in 1818, contains a reference to Girtanner's report of Rush's article on cholera infantum. "The disease occurs in Germany for the most part only sporadically, and especially when the teeth are breaking through the gums. However, it is seen at times also frequently, especially when after great summer heat diarrhœa and dysentery become epidemic in the fall." The fourth edition of this handbook, published in 1837, contains the identical article on cholera infantum as the first edition, without any new matter. Jahn's *Ueber Kinderkrankheiten*, Arnstadt, 1803, only mentions diarrhœa as being among other diseases of

frequent occurrence in children, and Wendt's *Die Kinderkrankheiten*, Breslau, 1835, a book of 470 pages, has nothing to say about diarrhœa. Meissner's *Die Kinderkrankheiten*, (Leipzig, 1838,) speaks of cholera as a disease of children occurring epidemically in America, but mostly sporadically in Germany; although it sometimes approaches epidemic proportions as a diarrhœa and dysentery together, when the nights are cold after hot days. Bressler's *Die Kinderkrankheiten*, (Berlin, 1842,) contains a chapter on cholera infantum, composed chiefly of quotations from American writers. The author still speaks of it as a sporadic disease in Germany.

The French physicians were later than their German neighbors in recognizing cholera infantum. Capuron's book on diseases of children, (Paris, 1821,) mentions the frequency of diarrhœal affections in infants, "to which they are predisposed by natural weakness and sensibility," and gives as exciting cause of these disorders, cold and moisture, and improper food. Nothing is said of the effect of atmospheric heat, or of special prevalence of the disease at any one season of the year. Billard, in the third edition of his book on diseases of children, (Paris, 1840,) recognizes the importance of intestinal disorders in infants, and devotes fifty pages to the discussion of the different forms. Under the heading of cholera, he says: "Cholera is not generally noted in our climate; it is a disease peculiar to infants in the United States; and as I have not observed anything analogous to this affection, I must be indebted to Dr. Dewees for the principal details of the symptoms."

The physicians of Great Britain were also slow in recognizing cholera infantum, and it was not until the middle of the Nineteenth Century that the disease was properly appreciated by them. According to the account given by Dr. John Cheyne, this omission was not due to any lack of prevalence of the disease, but to the indifference of the physicians and their failure to observe it. Cheyne describes in his "Essays on the Diseases of Children," published in Edinburgh in 1801, in the chapter on Bowel Complaints, affections which appear to be analogous to cholera infantum, and which he designated as "green scours" and "atrophia ablactatorum" or "weaning brash." He reports a case of green scours in a child nine months old, who was taken sick on the 10th of June, which we would not hesitate to consider an instance of cholera infantum. "This disease is sup-

posed to be caused by diet offensive to the stomach, or cold." He considers "weaning brash" one of the most fatal of the diseases of children, and states that it had been overlooked by those physicians who had made these diseases their study. As evidence of its fatality, he mentions that he had lost seven cases of it in one season. He does not associate the disease in any manner with atmospheric heat, and gives no evidence of acquaintance with the description of cholera infantum by American writers. He holds it to be "an undescribed disease belonging to the autumnal season, seldom occurring before the solstice or after the end of the year. It is an atrophy, the consequence of weaning children too suddenly at an unfavorable season of the year. It sometimes comes on in two or three days after weaning; frequently not for three or four weeks; sometimes not before five or six weeks have elapsed. It begins with purging and griping pains, and after continuing for some time, there is added a retching with or without vomiting." These studies of Cheyne are exceedingly valuable and represent the first original work done on this subject in Europe. His book contains reports of a number of cases of weaning brash and of post-mortem examinations. In most of the cases reported the sickness began in the summer months. They seem to have resembled the cases of cholera infantum encountered by us, which drag along through the summer, the patients beginning to improve or dying when cool weather sets in. He found in the autopsies "the intestine generally pale and without evidence of inflammation; mesenteric glands enlarged and red; liver exceedingly firm, larger than natural, and of a bright red color." The experience thus gained led him to believe that the mesenteric enlargement was an effect and not, as had been supposed, the cause of the disease; and that weaning brash in its confirmed form is imputable to an increased secretion of acid bile, or rather to the morbid state of the liver which occasions this. This condition of the liver he attributes to irritation of the intestine caused by improper food.

In the treatment of the disease he believed that the success of remedies depended upon strict attention to diet. His knowledge of dietetics, indeed, was far in advance of his time, and the rules laid down by him are far more appropriate than those recommended by the American physicians in the treatment of cholera infantum. He allowed animal broths, juice of lean

meat, milk and eggs, and particularly recommended thin rice-water or barley-water with a small proportion of skim-milk as a very proper drink for children. He found calomel very useful, and used it in half-grain doses, morning and evening, and says that he had much better success after adopting this remedy.

The second edition of Underwood's "Treatise on Diseases of Children," published in London in 1789, contains a chapter on diarrhœa, in which he says, "Diarrhœa is a complaint often as difficult to treat as any in the infant state. Lientery, or true watery gripes, is esteemed the most dangerous of all purges. Vomiting and purging often exist together, and they frequently arise from unwholesome milk or other food, and from moist, cold air." He makes no reference to any greater prevalence of these affections in the summer months, nor does he show that he had any knowledge of cholera infantum. The third edition of this book, published in 1815, has a similar article on diarrhœa and contains no reference to the American disease. Armstrong, in 1792, described a diarrhœa in infants which resembles cholera infantum, but he did not recognize it as such. Herdman's "On the Management of Infants," London, 1807, contains nothing on diarrhœa except a short quotation from Underwood.

Burns in his work on "Diseases of Women and Children," published in London in 1814, recognizes the importance of diarrhœal affections in infants. He considered purging to be a disease of the utmost importance, and one that should be early attended to. "It annually carries off many children, some of them suddenly, most of them after a lingering illness." No mention is made of any relation of the diarrhœa to a particular season of the year or to the cholera infantum of America.

The "Treatise on Diseases of Children," by Evanson and Maunsell, published in Dublin in 1836, has a chapter on cholera infantum which gives the earliest description of its recognition in Great Britain that I have been able to find. It is described as a disease of a very formidable character, occurring in the autumnal months as an epidemic, like other bowel complaints prevailing at this season.

The authenticated history of the summer diarrhœas of infants begins with the article on "An Inquiry into the Cause and Cure of The Cholera Infantum," written by Benjamin Rush, in 1773, and published in the second volume of his "Medical Inquiries and Observations." The additions to this history up to

the middle of the Nineteenth Century have been made exclusively by American writers: and if we have to regret the paucity in the number of papers written on the subject within that time, we find compensation in the value of those which have been handed down to us. Among those who recognized the importance of cholera infantum in those early days, and who have left us an account of their experience on this subject, are some of the ablest medical men this country has produced. So thorough and careful were the observations which they made, that apart from the lessons taught by the new science of bacteriology, the latter half of the Nineteenth Century has added but little to what was known on the subject in the first half. Dr. Rush's paper gives a description of the stools, the symptoms of cholera infantum, and a discussion of its etiology and treatment. It is only six pages long, and has not a superfluous word in it. It is remarkable for the amount of information it contains, and for the accurate description of the clinical features of the disease. He denies the etiological importance which, up to that time, had been ascribed to such factors as dentition, worms and summer fruits, and gives his views as to the nature of the disease as follows: "From the discharge of bile which generally introduces the disease, from the remissions and exacerbations of the fever which accompanies it, and from its occurring nearly in the same season with the cholera and remitting fever in adults, I am disposed to consider it as a modification of the same disease. Its appearance earlier in the season than the cholera and remitting fever in adults, must be ascribed to the constitution of children being more predisposed from weakness to be acted upon by the remote causes which produce those diseases." He does not attempt to explain any relation of the disease to atmospheric heat, and contents himself with saying that "the disease seldom appears in Philadelphia till the middle of June, and generally continues till near the middle of September; and its frequency and danger are always in proportion to the heat of the weather."

In the treatment he considered that the first indication was to evacuate the offending contents from the stomach and bowels. For the former he advised gentle doses of ipecacuanha or tartar emetic, and for the latter calomel, manna, castor oil or magnesia. "When there is reason to believe that the offending contents have been discharged by

nature, emetics and purges should by no means be given; but instead of them, opiates should be administered to quiet the stomach and bowels and to relieve pain." He also advised demulcent drinks; clysters of flaxseed tea or starch solution with a few drops of laudanum; warm applications to the abdomen, and warm baths. As soon as the more violent symptoms of the disease were composed he ordered tonics and cordial medicines, especially decoction of bark and port wine. His dietetic treatment would be considered exceedingly bold from a modern standpoint. He says, "After the disease has continued for some time, we often see an appetite suddenly awakened for articles of diet of a stimulating nature. I have seen many children recover from being gratified in an inclination to eat salted fish, and the different kinds of salted meat. In some instances they discover an appetite for butter and the richest gravies of roasted meats, and eat them with obvious relief to all their symptoms. I once saw a child sixteen months old, perfectly restored from the lowest stage of the disease by eating large quantities of rancid English cheese and drinking two or three glasses of port wine every day. She would in no instance eat bread with the cheese, nor taste the wine if it was mixed with water. After all that has been said in favor of the medicines above mentioned we have often seen them administered without effect." His principal dependence for many years had been placed in country air. Out of many hundred children sent into the country in every stage of the disease he had lost but three. He advised that they be carried out on horseback or in a carriage every day. As a prophylactic he recommended the removal of children to the country before the approach of hot weather, and cleanliness both with respect to the skin and clothing. He concludes his views on treatment with the statement that cholera infantum sometimes assumes symptoms of such malignity as to require bleeding to cure it. In some cases he says two or three bleedings were necessary for that purpose.

Twenty years after the appearance of this first paper on cholera infantum, Dr. Ed. Miller contributed the second article entitled, "Remarks on the Cholera or Bilious Diarrhœa of Infants," which was published in the *Medical Repository*, Vol. I., 1798. This is a practical paper, one of the principal objects of which is to recommend the trial of mercury alone, or with

opium, as different states and exigencies of the disorder may require. He says, "Calomel not only possesses special advantages as a purgative, but also other qualities of value in the relief of this disease, such as facility of exhibition, the difficulty of dislodging it from the stomach by the most violent vomiting; by its combination with opium much more of each ingredient can be safely and advantageously given than in a separate state; it is calculated to obviate the most fatal tendencies of the disease such as effusion in the head, or destruction of the organization of the stomach and bowels. Calomel when combined with opium, and especially when exhibited in small doses, excites a strong absorbent action, with respect to the fluids poured into the stomach and bowels. Calomel, though commonly safe and gentle, is the most penetrating, detrusive, and effectual of all the means employed to cleanse the intestinal canal; it dislodges substances not to be moved by other purgatives; and often discharges more bilious and other acrid matter of every description at one, than other cathartics at several evacuations. As to the dose of the medicine and the mode of exhibiting it, it is sufficiently explicit to observe that from ⅛ to 1 grain combined with $\frac{1}{10}$ to ½ grain of opium repeated every second, fourth or eighth hour, will comprise all the range of variety necessary in the treatment of the disease." Dr. Miller also advised careful attention to the skin and particularly cold baths in fever, also enemata of cold water. In the advanced stages of the disease he considered alum very valuable. He believed, with Dr. Rush, in the malarial origin of the disease.

Dr. James Mann, of Boston, wrote "A Dissertation on Cholera Infantum"* in 1803, which was published in pamphlet form, and for which he was awarded the Boylstonian prize. He considered all the varieties of complaints of the alimentary canal with which children are epidemically attacked in the months of summer and autumn, however differently modified in external appearances, as radically constituting only one disease, originating from a common cause, and requiring the same general remedies. He believed the disease originated from an infectious state of the atmosphere which was derived from filth and dirt, combined with heat and moisture. "The

*This dissertation was reviewed at length in Vol. viii. of the *Medical Repository* and it is from this review that the present account is taken.

characteristic principles of these subtle materials elude our researches. They have been variously denominated according to what has been supposed their nature and origin, morbific effluvea, marsh miasmata, mephitic vapor, animalcule contagion, putrid fermentation, nitrous acid gas or septan. It is probable that they may not be dissimilar to those offensive agents which are generated within the alimentary canal from acetous and putrid fermentation. These principles of infection must always exist in the common atmosphere, but become active only when they are in a concentrated state. This is one reason for greater prevalence of the disease in cities and towns. If the preceding statements are just, heat will be considered as one remote cause of cholera infantum, a torpor of the liver induced by the excessive excitement of that active agent as an intermediate cause, and a superabundance of acids in the alimentary canal in consequence of defect of bile as an immediate or proximate cause."

In the treatment of the disease Dr. Mann claimed to be very successful. He used mild emetics and cathartics to remove offensive material from the alimentary canal, and for this he preferred ipecacuanha and calomel. He attached special importance to correcting acidity with a weak solution of carbonate of potash or soda, repeated every two or three hours. "If inflammation has taken place, this should be allayed by giving a full dose of calomel and then administering it in small doses with or without opium. When the diarrhœa is accompanied with tenesmus, calomel combined with opium seems to possess specific property. In cases of violent vomiting and purging, commonly called cholera infantum, emetics and cathartics are not proper. A full dose of solution of alkali should be administered, and repeated as often as it is rejected. If vomiting continues, add to the alkali one or two drops of laudanum or embrocate the stomach with the strongest laudanum." He also used clysters with laudanum and clysters of cold water, applied blisters to the back, and smeared the skin all over with oil. He says in more than one hundred cases of the several forms of diarrhœa treated according to the above plan, only one patient had died.

In 1812 Dr. James Jackson, of Boston, contributed in the *New England Medical and Surgical Journal* one of the most complete and valuable articles that has ever been written on cholera infantum. He says, "The disorder is rarely seen in

Boston before the month of August, and it grows more frequently and is commonly more severe in September than in August. In most cases it is preceded by the diarrhœa of teething children. In its most exquisite form it is clearly distinguished from the diarrhœa; but there are many cases which are intermediate." He gives an accurate description of the stools and of the symptoms of the disease. He says, "In the protracted form of the complaint, the child lies asleep in its crib, cold amid the load of woolen in which he is wrapped, unless during a febrile paroxysm, when an acrid warmth is spread over him; his limbs so peculiarly dead in their appearance that it would seem that life was preserved only in its sacred temple in this 'little world'; his countenance more than deathly, and with which the visage of pulmonary consumption will scarcely compare; his pulses quick and wiry, and his respiration scarcely to be heard. So strongly, under these circumstances, are the characters of death impressed on the little subject, that the inexperienced observer cannot doubt that a few hours will decide the case forever. That the appearances have been the same for days and even sometimes for weeks, seems to be impossible. But the patient is wonderfully altered when he wakes. There is not perhaps any case where the distinction of animal and organic life, and their nearly independent existence, is more strongly marked. His clear eye seems to view the objects around him with peculiar intelligence. With the utmost decision, he chooses the pleasant and rejects the offensive things which are offered to him. He seems almost to tell you, by his actions, that his stomach is faint, and sinking, and distressed; that the call for something to support it is most painfully imperious; but that he can scarcely find an article which does not disgust it. The child is not disposed to make exertion, but when he does, there is often displayed a momentary energy of will, altogether disproportioned to the other appearances about him. In this situation the patient may continue for weeks, with some fluctuations, and at length recover. When death ensues to the feeble state, which has been described, the patient shrinks still more, and the countenance grows yet more sharp and hollow; or it becomes bloated, and has a fulness which deceives only the most careless observer. The feeble powers of life are at length surrendered, when they can scarcely allow to death the glory of a triumph."

The pathology is enriched by a number of autopsies which

were made in common by Dr. J. C. Warren and Dr. Jackson, during a period of several years. These autopsies are the first which have been reported on cholera infantum, and are of great value in establishing the inflammatory character of the disease. The record says, "The body is emaciated, often very much. In every case marks of disease have been found in the mucous membrane of the stomach and intestine. In the stomach there have usually been observed one or two spots of an irregular shape, in which the mucous membrane was red, inclining a little to purple, and the mucous membrane is commonly lined with an adhesive mucus. In the duodenum there have been found invariably one or more spots much larger than in the stomach, in which the mucous membrane has been considerably inflamed, and for the most part swollen. In almost every case such an inflamed patch has been found at the very commencement of the duodenum. In other parts of the small intestine other such inflamed portions of the same membrane have been seen in every case. In the large intestine it is rare to discover marks of disease; but such have sometimes been discovered, and particularly where dysenteric symptoms had existed. In one case there were strong marks of inflammation of the membrane throughout the large intestine, and frequent small ulcerations, resembling the canker spots in the mouth. The organs of the thorax, and the other organs of the abdominal cavity presented a normal appearance, excepting the liver, which was enlarged in some cases, but otherwise seemed healthy. The contents of the cranium were not examined. It is to this inflammation of the mucous membrane of the stomach and small intestine that the peculiar phenomena of cholera infantum may be traced in the same manner as those of dysentery have been found to be owing to a similar affection of the large intestine. It is when this inflammation supervenes in the autumnal diarrhœa of infants that the disease assumes its serious and threatening aspect; and it is at this time that the popular remark is made, that now the 'canker has seized the bowels.' This inflammation is no doubt much more severe and extensive during life, than it is found to be after a slow and lingering death."

Jackson attributed the chemical changes which take place in the contents of the stomach and bowels "to putrefaction of animal food, and the acetous fermentation of that

which is vegetable." He summarizes the legitimate effects of the inflammation as follows: "1st. The sensibility and the irritability of the parts will be augmented. 2d. The parts will cease to perform their proper or peculiar functions, or perform them with difficulty. 3d. The parts will secrete fluids different in quantity and quality from those secreted in health. 4th. The organs affected will be in some measure incapable strongly and vigorously to propel their contents; while in consequence of the increase in their irritability and sensibility they will frequently be excited to painful efforts to this purpose. 5th. Organs subsidiary to those immediately affected will have their secretions altered in quality and quantity. 6th. The constitution will be affected by sympathy. and the whole system will suffer, though not so immediately, from want of nourishment." He considered the principal remote causes of the disease to be, dentition, the season, improper food, restraint from exercise in the open air, and an impure atmosphere. Under the head of season, he says, "It is evident that there must be some cause operating in the summer and autumnal months favorable to the production of this disease. Whether there is one or whether there are various causes belonging to this season, to which its various diseases may be attributed, we do not undertake to consider. Nor shall we inquire in what manner the season operates in producing any of the causes of disease."

In the treatment of cholera infantum he considered it of first importance to remove all noxious matter from the stomach and intestine, and through the whole course of the disease to prevent the undue retention of fecal material. Calomel in doses of four to six grains he considered best for this purpose. He used opium to lessen the irritability of the intestine, and gave careful direction for the use of this remedy, and against its abuse. He recognized that milk was not always digested, and when it disagreed, he advised decoction of animal broths. and juice expressed from broiled beef. When the stomach was very irritable he gave very small quantities of liquid, a teaspoonful at a time.

Dr. C. D. Meigs, in an article "On Cholera Infantum or Summer Complaint of Children," published in the *American Medical Recorder*, 1820, advanced the congestion theory of the disease, and held dentition to be one of the most important

etiological factors concerned. He says "the cutting of the teeth in all seasons is commonly attended by symptoms which characterize in some degree the cholera infantum, such as, marks of an imperfect or vitiated digestion, restlessless, fretfulness, thirst, cough, cutaneous eruptions, and commonly more or less fever. All these circumstances, in connection with the vitiated atmosphere of a populous city, in the hot season, are sufficient to lay the foundation of a disease which exerts such a fatal sway. Increased excitement destroys the equilibrium of the circulation, producing a determination towards the larger trunks of vessels. In children these directions are principally towards the brain or hepatic veins. Continued atmospheric heat exhausts the tone of the skin from the incessant demands on its great and important function of perspiration. The skin becomes dry and dusky, and no longer performs with regularity its important share of elimination; the kidneys are not rendered more active, but on the contrary less so, and hence the alimentary canal becomes diseased by a load of crude matter rapidly and, therefore, imperfectly secreted. It would seem that in violent cases the portal system is so charged with blood, that a complete apoplexy exists in the liver, and that the organ is so oppressed by this condition as to be unable to relieve itself by a free secretion." He believed that cholera infantum depends principally on a loss of the healthy function of the liver, and that most of the collateral symptoms may be traced to the prevailing influence exercised by it over the other organs of the body; that the disorder arises not from an organic change in the structure of the organ, but probably from a congested state of the whole system of the vena portæ.

Dr. Meigs was very conservative in his treatment of cholera infantum, and deprecated the doctrine of purgatives which was so universally applied in his day, although he considered it necessary to keep the bowels free. His chief dependence was placed upon such measures as are calculated to restore the lost or vitiated secretion of the liver. For this he relied upon calomel in $\frac{1}{15}$ grain doses with an equal proportion of opium and ipecacuanha. This combination carefully administered every hour, or every two, three, or four hours, he says, rarely failed to bring down a free secretion of yellow bile, and was often followed by the happiest effects. He believed that calomel had a specific relation with the liver, by exciting in it a grade of

action compatible with its mode of vitality, and that when properly administered its exhibition is followed by an increase of biliary secretion, which is generally of a better quality, and as this stimulates the intestine, it improves digestion, while its flow relieves the congested vessels. In his experience a copious bilious stool was almost always followed by an improvement in the condition of the patient. He gave calomel, only in small doses, for its alterative effects, castor oil with laudanum to procure the regular evacuations of the bowels, and vegetable astringents to restrain their excessive movements. He paid strict attention to clothing and diet, and his advice in respect to both of these matters is excellent. He considered gum-arabic with lime-water and new milk, or preparations of arrow-root, sago, or tapioca with a proper admixture of milk, to be the most suitable diet for this disease. After all, in his opinion, no plan of treatment could stand in competition with that of sending children to the country.

Dr. E. Howell, of Philadelphia, has an article on "Cholera Infantum" in the *Medical Recorder* for 1823, in which special stress is laid on atmospheric heat in the causation of cholera infantum. He says, "Whatever consideration attaches to irregularities in diet, inattention to cleanliness, difficulty of dentition, etc., I am disposed to consider the violent heats of the summer in conjunction with sudden aerial vicissitudes, or with exposure to a moist and vitiated atmosphere, as the most usual exciting cause of the complaint. Infancy is the season of sickness. The various modes of vital action, not as yet strengthened and confirmed by habit, become susceptible of rapid changes from otherwise slight causes. The digestive system, in particular, from having an excessive function to perform in building up the corporeal frame, is liable to frequent derangement, particularly during the development of the primordial teeth. Vacillations occurring between the cutaneous secretions on the one hand, and that of the digestive canal on the other from frequent and sudden changes of temperature, as they render the system more accessible to the impressions of external agents, so they diminish the capacity of resistance to the operation of their noxious qualities. As the tone and energy of the digestive organs under the increased action of the cutaneous vessels become impaired, while its irritability under the powerful impression of heat is increased, it is thus more readily disposed, especially when

subjected to the concurrent influences of some of the above causes, to take on inflammatory action." He believed cholera infantum and the bilious or remittent fevers were produced by the same cause, viz., sudden vicissitudes of temperature accompanied with moisture. He shows an intelligent acquaintance with the pathological lesions of the disease, and mentioned those usually found in post-mortem examinations, but does not tell us by whom these examinations were made. He mentions a post-mortem condition which had not previously been noticed, as follows: "So trifling are the marks of preceding inflammation that we are at a loss to what circumstance to attribute the death of our patient, except to excessive exhaustion, or a peculiar state of the cerebral organs."

The first account of cholera infantum in the Southern States is found in a little book written by Dr. George Logan, of Charleston, S. C., in 1825, entitled "Practical Observations on Diseases of Children." Dr. Logan tells us that when cholera infantum first appeared it was of a much more serious character than it had become at the time of his writing. "The mortality among infants from this cause, anterior to the last thirty years, was awful and distressing; and it continues to occur, but by no means so general and severe as at those periods. It cannot fail to excite the most pleasing and grateful emotions, to observe that this evil does not increase, but very sensibly diminishes. Previous to 1820, Reports of Health exhibited the death of a larger portion by one-fifth, than has been subsequently known. An increased salubrity of climate, which accurate records, faithfully preserved for many years, incontestably prove, has greatly diminished this source of anxiety. The late Dr. Ramsey, in his valuable Medical History of South Carolina remarks prior to 1800, that the spring months were formerly the terror of parents, and that diseases of that season had become less frequent and less mortal."

Logan attributed the disease to "pain and irritation of teething, season of the year, neglect of cleanliness, and local circumstances. These concur in producing an increased and vitiated state of bilious secretion, acrimony and other disturbances in the first passages."

Dr. D. F. Condie made a valuable contribution to the treatment of cholera infantum in an article on the "Pathology and Treatment of Cholera Infantum," in the *Philadelphia Journal of*

Medical and Physical Sciences for 1825. He, however, said but little on the pathology of the disease, although he appreciated the importance of this part of the subject, as is indicated by the following sentence: "Had only a portion of that time and those talents which have been wasted on useless inquiries into the cause of life and the essential nature of fever, been spent in carefully investigating the pathology and method of treating cholera infantum and other diseases, which, though of frequent occurrence, are but little understood, it would no doubt have been the means of preserving, not individuals, but thousands and tens of thousands from a premature grave." He believed the disease to be a mere variety of the bilious fever of our climate, "the force of which is turned inwards upon the intestines," and that it owed its origin to an overheated and vitiated state of the atmosphere. "By constant exposure to excessive heat, particularly when from any cause the surrounding air has become vitiated or loaded with miasmatic exhalations, a debility of the cutaneous vessels is produced by which they are rendered unequal to the circulation of their accustomed fluids; the consequence is, perspiration is diminished, and, at length entirely suspended, while the circulating mass driven from the surface, accumulates progressively in the central trunks; the vessels of the internal viscera become morbidly distended by the influx of blood; the lining coats of the stomach and bowels acquire a high degree of irritability, and their secretions are much increased and changed in quality, whence they become irritating, and excite the prominent symptoms of the disease. The surrounding organs participate in this irritation, resulting in functional derangement with suspended or vitiated secretion."

Dr. Condie followed the purgative plan of treating cholera infantum, and he makes a statement on this subject which I have not seen before: "There is a prejudice with many practitioners, though it is happily fast wearing away, against the use of any purgative, but particularly calomel, in cholera infantum." He preferred calomel as a cathartic to procure bilious evacuations, and generally gave it in combination with ipecacuanha. He found that as soon as a copious bilious stool was produced, all the symptoms of the case were ameliorated. He disapproved of emetics, and of opium and astringents. If nature needed assistance in removing offensive matter from the stom-

ach, he employed diluent drinks, and thought that he had obtained good results from the use of spirits of turpentine in ten to thirty drop doses, in quieting the violent action of the stomach. He believed bleeding to be necessary under certain circumstances. For diet he recommended mother's milk, decoction of arrow-root, sago or rice, in milk, and fresh whey. Like Dr. Rush he had seen cases benefited by the use of salted meats.

Dr. Dewees in his "Treatise on the Physical and Medical Treatment of the Diseases of Children," published in Philadelphia in 1825, devotes a chapter to cholera infantum, which gives a good history of the disease, and excellent recommendations for its treatment. He does not discuss the remote etiology, and gives as the chief exciting causes, improper diet and clothing. His views on the pathology of the disease appear to have been derived from Dr. Jackson's work. In the treatment of cholera infantum he disapproved of emetics and purgatives for evacuating the stomach and bowels. For this purpose he had found nothing so certain or so prompt as an injection into the bowels of a gill of warm water, in which is dissolved a large teaspoonful of common salt. He says, "If it operates immediately and brings with it a fecal or bilious discharge the stomach becomes almost immediately quieted. This injection should be repeated whenever the vomiting is severe. If it should fail to quiet the stomach, draughts of warm water or even cold water, where the warm will not be drunk, or strong coffee in teaspoonful doses every fifteen minutes, may be given." If these measures failed to tranquillize the stomach, he resorted to calomel in doses of a half, a quarter, or an eighth of a grain. Dr. Dewees was one of the first to employ calomel in this disease in small doses, as he began the use of it in this manner in 1795, and he was severely ridiculed by some of his medical friends for the supposed insufficiency of the dose. In the later stage of the disease he used vegetable astringents, opium, sugar of lead, and chalk mixtures. For diet he preferred breast-milk, but he recommended that care should be exercised in giving the breast, not to allow the child to suck too much, nor too often, especially if thirst is excessive. When the child would not take the breast, he used milk diluted with barley-water, rice-water, or gum-arabic tea. The only remedy he regarded as sovereign and nearly unfailing was a change of air.

Cholera infantum extended rapidly with the advance of immigration into the interior of this country, and in the third decade of the last century several interesting papers on the subject were written by physicians in the western states. In 1826 Dr. Samuel A. Cartwright, of Natchez, Miss., published an essay on the "Pathology and Treatment of Cholera Infantum," which appeared in the *Medical Recorder*, Vol. X, and for which he was awarded a prize by the Medical and Chirurgical Faculty of Maryland. He states that the disease prevailed to an alarming extent in Cincinnati, being particularly severe in 1819. His paper contains one of the most complete and accurate accounts of the clinical features of the disease to be found in the early literature. Dr. Cartwright was also the first observer who noted the frequent appearance of a cough when the disease has passed to an inflammation of the intestine. His views on the pathology are novel and purely theoretical. He divides the disease according to seven different pathological states of the system, as follows: 1, Visceral congestion; 2, congestion with an ataxic or broken reaction; 3, congestion with a general reaction or fever; 4, local inflammation with general reaction or fever; 5, local inflammation, with an ataxic reaction, or broken or partial fever; 6, local inflammation unattended with reaction; 7, local inflammation, terminating in effusions, or in structural derangement of some viscus. He says that in some cases one or more of these states are entirely absent. He adapts a special treatment to each of these conditions, and describes under each head the distinctive phenomena to which they give rise. He believes in the miasmatic origin of cholera infantum, and that the miasm enters the body through the air-passages and affects "the pneumoganglionic nerves, which are interwoven in the extensive mucous membrane of the lungs, producing a morbid impression which interrupts in some measure the arterialization of the blood. The miasmata applied to the extremities of the nerves in the lungs, during the respiration of the atmosphere of an infected district, produce a morbid impression which is conveyed to the ganglion, the centers of the nerves of organic life, and there produce disease. All those parts which are supplied with nerves from the ganglions in the liver, the stomach, the bowels, etc., become diseased in consequence of the diseased state of the ganglions, and their plexuses from which they derive their nerves." This opinion was founded

upon the diseased condition of the ganglions and their primitive plexuses which he had found in numerous post-mortem examinations in miasmatic disease.

Dr. Cartwright devotes seventeen pages of his paper to the treatment of the disease, and though he advocates the use of many remedies in heroic doses, his careful study of each patient and special adaptation of the treatment to the particular condition of the system is interesting and instructive. He had an abiding faith in tartar emetic in certain stages of the disease, and gave it in half-grain doses every two hours, believing that in the appropriate stage this amount is not so liable to produce vomiting as the smaller dose. He gave calomel in four grain doses to a child in the second summer, and never less than three to five grains to a child no matter how young. "In these doses calomel not only excites the liver, but other organs, as the kidneys and skin, to action, and it acts on the bowels more certainly, more readily, and less copiously than the smaller dose. He preferred croton oil to castor oil for cleansing the bowels, and gave it in doses of one-third of a drop. For the exhausting discharges he says the greatest benefits are derived from a combination of extract of white walnut, sugar of lead and opium, which had been recommended by Dr. Henderson, who treated this disease with unparalleled success. He considered blood-letting often necessary to quell the arterial commotion, and preferred local to general blood-letting. Why half the quantity of blood taken away by leeches has a better effect in abating arterial reaction than twice the quantity drawn by the lancet, he did not know. He recommended warm and tepid baths, and cold sponging in fever, and for diet, vegetable broths when breast-milk was not available.

In 1828, in the *Transylvania Medical Journal*, Dr. John P. Harrison gave an account of cholera infantum as it appeared in Louisville, Ky. He considered atmospheric heat to be the general predisposing or remote cause of the disease, and improper diet the most important exciting cause. He thought that dentition might aggravate the disease when existing, and might even act as an exciting irritant in developing it, but considered it only as an adventitious circumstance. He explained the mode in which heat predisposes to intestinal derangement as follows: "There is an intimate relationship of function between the stomach with its dependent organs, and the surface. In

cold weather the power of digestion is increased by the tone imparted to the stomach from the tonic influence of moderate cold on the skin. In hot weather there is a diminution in the power of the stomach produced by the action of heat on the skin." This paper gives a good description of the symptoms of the disease, and valuable directions for its treatment. The indications recognized are: to remove the existing cause; to restore the natural functional action of the stomach, bowels, liver and skin; to sustain, when restored, such healthy action. For emptying the stomach he preferred ipecac to tartar emetic, as he considered the latter too severe for children, and stated that several deaths had been supposed to result from its depressing effects. He advocated the use of fractional doses of calomel as the safest practice in the majority of cases.

The same volume of the *Transylvania Medical Journal* contains "An Essay on Cholera Infantum," by Dr. John Esten Cooke, of Transylvania University, which gives an able discussion of the etiology of the disease. He says, "Although hot weather is the season in which cholera infantum is epidemic, and heat is essential to the prevalence of this epidemic, and, therefore, to the existence of the cause of it, it is not itself the cause. The opinion that heat alone is the cause of cholera infantum has derived its chief support from the authority of Rush, who says its frequency and danger is always in proportion to the heat of the weather. This does not always hold good as is shown by the observations of Cleghorn in the Island of Minorca, and by my own experience in Virginia in the summer of 1821, which was uncommonly hot and dry, and nevertheless uncommonly healthy. There is evidently something more than heat necessary to the production of the disease, and to discover what this is, it is necessary to consider the circumstances in which the disease appears in different situations." In this connection he comments as follows upon Cleghorn's table representing the state of the weather, and the diseases in each year from 1747 to 1749 inclusive. "These years differ not so much in heat as in moisture. Though the former differs but little, there is a great difference in the latter, and on comparing the different summers together, we find that the wettest were the most sickly. From these facts it is evident that cholera infantum depends on some cause arising out of this combination of heat and moisture, when this is considered in connection with the

additional facts, viz., that cholera and tertians prevail together in every year; that when one appears earlier the other does also; both in consequence of the earlier appearance of that combination of heat and moisture which produces them both; and lastly that when there is a considerable deficiency of moisture neither tertians nor cholera are very prevalent, notwithstanding the heat of the weather is extreme, it is very evident that they both depend on the same remote cause arising out of this combination of heat and moisture, viz., on miasmata. The nature of the internal derangement produced by this remote cause consists of accumulation of blood in the vena cava and its branches. The stomach is affected by the same accumulation, which causes a deficiency of the gastric secretion. Fermentation takes place in the stomach owing to deficiency of gastric juice, and vomiting and purging result from the action of the fermented mass on the stomach and bowels, as well as from the increased sensibility and irritability of the alimentary canal, proceeding from the same accumulation in all the interior veins and small arteries entering into them." He adds, "In accordance with this view of the pathology of the disease the main dependence for its cure is in purging, so managed as to produce evacuations from the liver, and continued daily until health is restored. When the discharges are thin, serous and profuse, it is important to procure, as speedily as possible, consistent discharges from the liver, and to continue them. Calomel is better adapted to the end in view than any known remedy; in five grain doses every twelve hours, oftener in severe cases. It almost invariably effects the desired alteration in the passages in twenty-four to forty-eight hours. It sometimes happens that calomel does not purge the patient; the thin discharges cease entirely but no others appear. In this case rhubarb, jalap or scammony in five grain doses must be added. The object is to get the discharges green or dark and when this is accomplished the calomel may be diminished or omitted. When the case is not urgent the calomel should be given at bed time, as it relieves discomfort and procures sleep. This course of treatment has been entirely successful for many years." He disapproved of emetics, and of opium, and allowed anything for diet that the child desired and could retain, but recommended chiefly milk and water.

The first contribution devoted exclusively to the pathologi-

cal study of cholera infantum was made by Dr. W. E. Horner, of the University of Pennsylvania, and was published in the *American Journal of the Medical Sciences* for 1828, under the title of "An Inquiry into the Anatomical Characters of Infantile Follicular Inflammation of the Gastro-intestinal Mucous Membrane, and into its Probable Identity with Cholera Infantum." This valuable work attracted considerable attention. It contained a detailed report of the autopsies made on five infants dead of follicular inflammation of the intestinal canal—the symptoms being those of cholera infantum—accompanied with four beautiful plates illustrating the enlarged and ulcerated follicles. He says: "In all of the cases the muciparous glands of the colon were enlarged so as to represent small grains of white sand sprinkled over the mucous membrane and about the size of millet-seed. There was in each a little depression of a darker color than the rest of the gland, and from its position at the apex was taken for the orifice of the gland. The muciparous glands of the small intestine were also tumid and irritated. In a few of these follicles in both the small and large intestines ulceration had begun to show itself." He concluded from this study that "cholera infantum is pathologically rather a follicular than an erythemoid inflammation; that it is rather a disease of the innumerable mucous glands or follicles, extended from one end to the other of the alimentary canal, than a common vascular or erythemoid inflammation." In one of the cases Dr. Horner observed that the bile in the jejunum was yellow, but green in the colon, and offers the following explanation: "We know that frequently in cholera the alvine discharges are in a state of fermentation, and are sour. Is this process confined to the colon? If so, the rationale is that the bile retains its natural character in the small intestines, but becomes green in the colon, from meeting with acescent matters made so by fermentation." He found the other organs of the abdominal cavity healthy, but did not examine the thoracic or cranial cavities.

Dr. Nathaniel Potter, of the University of Maryland, contributed an able and interesting article entitled "Observations on Cholera Infantum," to the *Baltimore Medical and Surgical Journal and Review* for 1833. He urged the necessity of understanding the remote cause of cholera infantum, not only as a means to elucidate its pathology, but also to indicate an easy

prophylaxis. He says: "No palpable cause can be assigned in early summer so obvious as the overwhelming influence of a high temperature upon the exquisitely sensitive nervous system of children. It is scarcely possible to resist the conclusion that the matter of heat gives the first impulse, and that it originates a peculiar train of symptoms in the relative condition of infancy and childhood for which no other cause can account. That cholera is more clearly defined here than in other countries is due to the character of our climate. It is not only more variable than any other, but the vicissitudes are more frequent and the changes of temperature greater and more sudden. The disparity between winter and summer shows a contrast to which there is no parallel in any other country. In addition to this we have no gradually opening spring, but a sudden influx of heat in the later vernal months, which constitutes a sudden transition to summer. In its country localities the disease has uniformly increased in the ratio of the insolation of the soil. The first settlements which were soonest cleared of woods and underwood, and therefore more exposed to solar heat, were the first seats of cholera infantum. It is well known that there are many farms that were formerly resorted to as places of refuge, which are now infected with cholera. In traversing the forests of Caroline County, in this State, in the summer of 1796, while the disease might be said to be epidemic in the old settlements, the new dwellings and solitary log houses, invested by a thick and rich foliage, presented, without an exception, children with blooming complexions, the emblems of health." Dr. Potter considered marsh effluvia to have no agency whatever in the causation of cholera infantum, and mentioned, among other causes to which the disease has been attributed, a deficiency of oxygen in the atmosphere, which rendered it physically impossible for the lungs to perform their office in oxygenating the blood. Some of the adherents to this visionary theory proposed to increase the quantity of vital air by artificial means, and were so carried away with this idea that they invented an apparatus for the purpose of manufacturing and diffusing oxygen in a more concentrated form through the apartments of children in the summer. He adds: "Great improvements have been made in the treatment of the disease, and under favorable circumstances it can generally be cured, and the successful management is always accomplished by a few simple

means. Removal to the country, notwithstanding its acknowledged advantage, is not entitled to the high regard which it justly acquired at an early period of our medical history. A hut surrounded by woods with only a yard and kitchen garden is the most eligible spot that can be selected as an asylum against the disease."

The importance of establishing the correct etiology of cholera infantum was appreciated by these authors from the first, but their views upon the subject varied widely; inasmuch as the disease had not been recognized in other countries, it was supposed that a new disorder had come into existence, originating with the settlement of this country, and as the various types were confined to children in the first two years of life, and to a special season of the year, the solution of the etiological problem was sought for in the local conditions peculiar to this country, in conditions peculiar to the age of infancy, and to the summer season, which might be operative in the causation of the disease.

At first two factors, the presence of worms and dentition, were held to be the main excitants. Against both, however, Dr. Rush brought forward strong arguments which, as regards the former, were so convincing that it was removed entirely from etiological consideration, although dentition still continued to be considered by many as of great importance in this connection. By Dr. Jackson it was thought to be one of the necessary causes of the disease, and Dr. Meigs, as late as 1820, classed it as one of the most important. Some writers, however, held the views expressed by Dr. Rush, that dentition might aggravate an already existing summer diarrhœa, but never induced one. They attributed the influence of age in the etiology of cholera infantum to the greater impressibility of infants owing to weakness of organization, the digestive organs being particularly liable to derangement at this age from the excessive function required of them in building up the rapidly growing body.

A causative relation was observed between the summer season and cholera infantum, but no explanation for this was attempted by the earliest writers on the subject, further than to say that heat may be considered as one of the remote causes, and that the frequency and danger of the disease were in proportion to the heat of the weather. In 1823 Dr. Howell gave greater prominence to heat as an etiological factor. He con-

sidered it in connection with sudden atmospheric changes, or a vitiated atmosphere, to be the most usual excitant of cholera infantum. Ten years later Dr. Potter declared that heat was almost the exclusive remote cause.

That the disorder was so much more clearly defined here than in other countries was referred to the character of our climate—to the frequency, severity, and suddenness of the changes of temperature, and to the sudden transition from winter to summer, without the intervention of a gradually opening spring. According to the generally accepted opinion, heat, dentition, improper food, and a changeable climate were not sufficient to account for an epidemic of cholera infantum. Something else was required, and this was suspected to be of an infectious nature. In the absence of any knowledge of bacteria in this connection, attention was directed to marsh miasm as the most probable agent. The discrepancies between cholera infantum and malarial fever are so apparent to us, that an identity or close relationship of these affections could not be suspected. But viewed in the light of the knowledge existing at that time, it is not surprising that cholera infantum should have been classed with the remitting fevers. Indeed the fact that the exciting cause was sought for among the infectious agents speaks strongly for the careful consideration given to the etiology of cholera infantum.

The mode of action of atmospheric heat in causing cholera infantum was the subject of considerable controversy, and various theories were advanced in explanation of it. These were, for the most part, based on the prevailing conception of the existence of a cutaneo-hepatic sympathy. By some the heat of summer was supposed to exhaust the tone of the skin, or to disturb its healthy action, and thus determine an undue quantity of blood into the abdominal viscera, causing a congestion of these organs. By others the disturbance of the intimate relation between the surface of the body and the digestive organs, caused by protracted heat, impaired the tone and energy of the stomach, rendered the system more accessible to impressions exercised by external agents, and diminished its resistance to them. The most important advancement in the interpretation of the action of heat was made by Dr. Cooke in 1828. He considered that hot weather was essential to the existence of the cause of cholera infantum, but was not itself

the cause. This opinion in connection with that expressed above corresponds with the modern conception of the subject.

The pathology of cholera infantum received less attention than its etiology, although the advantages to be derived from a clear understanding of its nature appeared to have been fully appreciated. Considerable importance was attached to the condition of the liver by many of the writers. The organ was supposed to be congested and greatly enlarged, and its healthy function impaired or lost. By some this disturbance was thought to constitute the principal trouble of cholera infantum, and to account for most of the symptoms. This opinion was purely theoretical, but it had such a firm hold on the minds of the physicians of that day, that it was not abandoned even after proof of its falsity had been adduced by anatomical investigation. More correct views of the pathology of cholera infantum were first obtained from the anatomical studies made by Dr. Jackson, and later from those of Dr. Horner. Dr. Jackson clearly demonstrated the inflammatory lesions in the stomach and intestine, to which he ascribed the phenomena peculiar to the disease, but this valuable work failed to attract the attention it deserved. Dr. Horner's studies exerted a much wider influence, and by some have been credited with laying the first correct foundation of the pathology of cholera infantum. This opinion, however, would do manifest injustice to Dr. Jackson, whose work preceded that of Dr. Horner by nearly twenty years. Moreover, although the observations made by Dr. Horner led to conclusions different from those of his predecessor, the divergence was due not to any lack of accuracy on the part of Dr. Jackson, but to the small number of autopsies made by each of these distinguished observers. Those of Dr. Jackson were confined to cases of catarrhal enteritis, which is now recognized as one anatomical form of summer diarrhœa, whereas those of Dr. Horner were confined to another form, ulcerative or follicular enteritis.

Many of the writers recognized the injurious influence produced in cholera infantum by acetous and putrefactive fermentation in the intestine, and by alteration in the quality and quantity of the normal secretions in the digestive tract.

The treatment of cholera infantum varied almost with the individual writer, and was based largely upon their conception of its nature. The purgative plan was employed by many, and

purgatives were given in heroic doses; by others this method of treatment was disapproved. Calomel was regarded as a most valuable remedy by all of the writers, without exception. It was given in large and in small doses, and was used both for its purgative action and its supposed efficacy in restoring the healthy function of the liver. Emetics were considered by some to be of great value, by others to be dangerous. Removal to the country was regarded as the most useful of all measures for the relief of cholera infantum, and in Dr. Rush's experience was sufficient in most cases to afford complete relief. Later, its beneficial effects were not so apparent. The dietetic treatment varied from cereal waters and milk whey and these in small quantities, to almost anything on the table and in unlimited quantities. There are few measures and remedies now in use for the relief of cholera infantum that have not been recommended by some of these writers. However numerous and opposing the different plans of treatment employed, good results were claimed by all of them.

I have now presented a brief summary of the more important literature on cholera infantum up to the fourth decade of the last century. The views of each of the authors on the etiology, pathology and treatment of cholera infantum have been given in greater detail than may be of interest to any one not specially engaged in the study of this affection. The clinical history of the disease as portrayed by these writers corresponds so closely with that in the text-books on pediatrics of the present day that no advantage could be derived from its repetition.

Bacteriology has given us clearer ideas on the etiology of cholera infantum. Improved methods for pathological investigation have enabled us to determine more accurately the nature of the lesions of the stomach and intestine, and extension of the changes to other organs of the body. Our treatment still leaves much to be desired, and perhaps our greatest triumphs are won by careful dieting and hygiene, which are still our main reliance for combating the disease, and what is still more important undoubtedly affords the best prophylaxis.

A STUDY OF 555 CASES OF SUMMER DIARRHEA AMONG THE OUT-PATIENT POOR.

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The 555 cases of summer diarrhea were treated at the out-patient department of the Babies' Hospital during the months of June, July, August and September. They were all from one class of society, the tenement poor.

AGE OF PATIENTS.—75 were under three months of age; 87 between three and six months; 150 between six and twelve months; 187 between one and two years; 51 were over two years of age.

PREVIOUS DIET.—Of those under six months of age, 24, or 14½ per cent., were breast-fed; 15 between six and twelve months, or 10 per cent., were breast-fed.

The mother's statement that the child was nourished entirely at the breast without other feeding must be taken with some allowance, as there are very few tenement breast-fed infants who are not given milk, soups, tea, etc.

But 20 were fed the proprietary foods, which means that the expense of the proprietary foods fortunately prevents their use among the tenement population; 59 were fed on condensed milk; 1 was fed on goat's milk; 472 were fed on a cow's milk diet entire or in part. These figures again remind us of the necessity of placing good milk within the reach of the poor.

Six per cent. only of all the patients were nursed.

THE DURATION OF ILLNESS WHEN FIRST SEEN.—In 157 the illness was of twenty-four hours' duration when the case was first seen; 197 had been ill from two to five days; 76 had been

ill from five to ten days, 111 had had diarrhea for over ten days, 11 of which had been ill for two weeks.

SEVERITY OF ILLNESS.—All were brought for treatment because of diarrhea and they showed all the degrees of severity that this number and class of patients might be expected to present during the summer months. With some at the first visit there was simple so-called dyspeptic diarrhea, from six to ten passages daily, with moderate fever and slight prostration, others had been ill for several days with high fever, prostration and emaciation and were in a critical condition.

DURATION OF ILLNESS UNDER TREATMENT.—80 cases recovered in three days; 168 in from four to seven days; 79 in from seven to twelve days; 62 in from twelve to seventeen days; 11 recovered during the third week; 58 during the fourth and fifth week; 11 during the sixth and seventh week; 6 from the eighth to the tenth week; 15 from the third to the fifth month.

In 56 the treatment was not continued; some failed to return and could not be traced. In a few further treatment was refused because of the failure of the mother to comply with directions.

499 were treated to a conclusion of the illness; of these, 10 died, the death-rate being a little over 2 per cent.

Among the fatal cases 1 had been ill twenty-four hours when first seen; 1, forty-eight hours; 3, one week; 2, two weeks; 3, three weeks.

In Greater New York several thousand infants die every year from summer diarrhea. Our work, as carried on at the out-patient department of the Babies' Hospital, among the class in whom this large mortality occurs, proves that the mortality is preventable and suggests a way for its relief.

In our management of summer diarrhea we have one invariable rule of treatment; *regardless* of the severity or duration of the illness, *regardless* of the diet, whether breast-fed or bottle-fed, whether the stools are frequent and watery, or infrequent and foul, we stop the milk at once. This is based upon the belief that in every case of summer diarrhea, no matter how mild, we have an infected gut, or soon will have it, and we wish to make the intestinal contents as poor a culture field as possible.

To the great mass of physicians it will always be impossible to differentiate bacteriologically the nature of the infection. Clini-

cally no one can differentiate with any degree of certainty. This has been impressed upon me by a considerable autopsy experience. At another institution I personally conducted 218 autopsies on children who died with summer diarrheas, and I have had the advantage of observing a great many other cases under treatment and later at the autopsy work of other physicians. Where we expected gross lesions we very often found them slight. In other cases which showed but little mucus during life and no blood, there were extensive ulcerations of the colon extending to the peritoneal coat. In a case which died last summer there was very high fever and intense prostration. A few yellow, well-digested, practically normal stools were passed twenty-four hours before death. Autopsy showed an extensive membranous colitis.

In the very acute cases in which death took place a few hours after the onset of the illness there was a pale, washed-out gut with perhaps slight enlargement of the lymph follicles. In spite of the uncertainties in diagnosis, we have profited by our experience. We have learned that every case of summer diarrhea must be looked upon as dangerous and treated vigorously whether the initial symptoms are mild or severe. *This we must teach.* A case of so-called dyspeptic diarrhea with milk feeding continued will soon become a virulent infection. I question if in the great majority of cases the streptococcus and colon bacillus play a very important part at the commencement of the illness. The average case is of gradual onset; we meet a few cases, however, in which the onset is sudden and severe.

We not only advise that the milk be discontinued, we command it, and if the order is not complied with further treatment is refused. To those of intelligence, among whom the American-born mother predominates, we explain why the milk should be discontinued for a few days. Our directions are almost always followed. The very ignorant, among whom the Italians, Russians, etc., figure largely, are given medicine and told that if the medicine and milk or any food except that which we advise, are given together they may kill the baby; the mothers give the medicine and feed the child according to our oral instructions which are supplemented by printed directions. No milk is allowed until the stools approximate the normal, which may mean an abstinence from forty-eight hours to five months; this was the longest time a child was kept on a non-milk diet. In three it could not be given for several

months. Fifty-seven suffered relapse upon resuming the milk diet.

If milk is discontinued a substitute must be offered. The milk substitute which we found most useful was a cereal water. As a rule barley water was used. When this did not answer rice water was given. Our directions were for Robinson's baked barley flour, two tablespoonfuls, water one pint. This is boiled for twenty minutes, strained and water added so that there is one pint when the cooking is completed.

Rice water is prepared by boiling two tablespoonfuls of rice in one pint of water for three hours, water being added as in preparing the barley, so that there will be one pint when the three hours' cooking is completed. With very few exceptions barley was the substitute selected. Additions in the form of liquid non-milk nourishment were added to change the taste and give the child a variation in the diet. A favorite mixture with us is four or five ounces of barley water and one or two ounces of broth—beef, mutton or chicken. Two teaspoonfuls of beef juice added to the cereal water often makes a suitable change. The taste of the substitute must vary or the child will soon tire of it. Broths must not be added in too large amount as in some a decidedly laxative effect will result. I usually order two or three substitute preparations and alternate them.

Brandy and whiskey, so frequently added to substitutes, should not be given to a child with summer diarrhea. The dangers of stomach involvement and nephritis are great without our provoking them by the use of alcohol. The white of egg mixture I have used rather extensively and discarded for the reason that many children fail to digest it and passing unchanged into the intestine it can form as good a putrefactive culture medium as milk. I look upon the brandy and egg water mixture, so popular with many, as an atrocious milk substitute. The cereal decoctions plain and dextrinized answer my purposes very satisfactorily. In the use of the carbohydrates the nature of the intestinal contents is changed from one of putrefaction to fermentation which does not furnish a favorable soil for the growth of dangerous pathogenic organisms.

Dextrinized gruels have a useful field in the diets for summer diarrhea. It is impossible to give a stronger barley water than two even tablespoonfuls to a pint of water for any length of time; twice this amount may be taken if the cereal is dex-

trinized. Four even tablespoonfuls of barley flour to a pint of water give a food strength of approximately .14 fat, .6 proteid, and 4. soluble carbohydrates. Dextrinizing is of value in that a more concentrated nourishment may be given the patient.

Care must be observed that the cereal water is not at a higher temperature than 100° F. when the digestive agent is added; if such is the case the dextrinized gruel will have a taste not unlike that of malt, to which the child will object. The substitute diet is allowed to be given at two hour intervals if the child will take and retain it in quantity that he was accustomed to take of milk in health. Boiled water is given at any time.

The mother is told to keep the patient in the largest room in the apartment. If there is fever she is instructed to give cold water spongings for fifteen minutes several times a day.

How is the milk diet to be resumed? It must be given gradually at the onset. I begin by using from one to four drachms with each feeding of the barley water, making a slight increase every day or every other day if the condition of the stools allow. The cereal gruel used as a diluent *does not* permit of a larger amount of milk being given at the commencement of the milk feeding, neither does its use permit of a more rapid increase in the milk strength than if plain water is used. I have demonstrated this in a great many cases.

DRUGS.—Various drugs have been tried from time to time. The newer tannin preparations were used in about 50 cases. Eudoxin in 20. These drugs were used in selected cases after the initial dose of castor oil and while under diet, and were continued until our medical conscience would not allow a continuation of the treatment.

During my term of four years' residence service at the New York Infant Asylum, salol, resorcin and almost every known drug that seemed to warrant a trial was given under Dr. Holt's directions to a series of cases. The year that followed I had the privilege of attendance upon the Clinics of Ranke and Zeitz in Munich and of Monti and Wiedehoefer in Vienna, where many cases of diarrhea were treated daily. During the eight years that have intervened in private practice, at the New York Polyclinic and at the Babies' Hospital, many hundreds of cases of summer diarrhea have been conscientiously watched and followed day by day. I have learned that there are just

four drugs that may be relied upon to serve us in summer diarrhea. These are calomel, castor oil, bismuth and opium. Either calomel or castor oil were given in every case. In 26 they were the only medication.

Calomel is preferred in a case in which there is vomiting or a tendency thereto and when the case is not particularly urgent, 1-20 to 1-10 of a grain is usually prescribed at hour intervals.

Castor oil is given in the acute septic cases with infrequent stools and without stomach involvement, in which a prompt washing out of the small intestine is desired.

Bismuth subnitrate was given in 432 of the cases treated. It was never given in less than ten grains every one or two of the waking hours, regardless of the age of the patient. When given persistently in this large amount it is a remedy of immense value. In order to be of service, however, it must produce black stools. In other words if some of it is not converted into the sulphid of bismuth in the intestine it is without value. If it passes through the bowel unchanged no influence whatever will be exerted upon the intestinal contents. This happens in a small percentage of cases and is explained in the absence of sulphureted hydrogen in the intestine, which condition is due doubtless to the absence of pancreatic digestion. In such cases the sulphur is supplied in the use of precipitated sulphur, a one grain powder being given with each dose of the bismuth, the bismuth is continued in the large doses until the child is ready for milk and then the dosage is diminished one-half and continued until full milk feeding is possible.

OPIUM.—The indications for the use of opium are pain, tenesmus and frequent stools. In a severe or even in an average case in which there is systemic poisoning evidenced by fever and considerable prostration, four or five passages a day are desirable. I look upon this number of stools as maintaining drainage. It was found necessary to use opium in connection with the bismuth in 200 of the cases. When the case is one of intestinal infection with infrequent foul stools or no stools at all, active laxatives comprise the only medication. Many children are brought to an untimely end because of the idea that the number of the discharges, that is, the diarrhea, must be stopped. The physician's efforts are directed to this end with little or no

attention to the diet and the nature of the intestinal contents, the cause of all the trouble.

IRRIGATION OF THE COLON.—As is the case with all good measures, irrigation of the colon has been overdone. Because a baby has diarrhea, it does not follow that he must be irrigated. A child who is having from ten to twenty loose, watery discharges in twenty-four hours is quite effectively washed out and does not require more. Time and again I have irrigated these cases and removed nothing whatever. The cases which are benefited by the washing are those who have a moderate number of green mucus stools with or without blood; in short, the cases to be washed out are those which have something to be removed. I never wash the colon oftener than once in eight hours, rarely as often as this; once in twelve hours accomplishes considerable and does no harm. Too frequent irrigation causes straining, distension of the lower bowel, fissures of the rectum, and, if not very carefully done, injury to the mucous membrane of the descending colon. Various solutions have been used for the irrigation. One is as good as the other if water enough is used, for it is the cleansing of the bowel which benefits the patient. I usually employ a normal salt solution. In case there is blood, a 1 per cent. solution of tannic acid is used. I question, however, if it is of any more value than normal salt solution. The solution selected is used lukewarm. If there is high fever I use it as cool as 60° or 70° F.; in the very weak, with subnormal temperature, with marked prostration, the solution is used at 110° F. Irrigation is carried on as follows: A soft-rubber catheter, no. 14 English, is attached to a fountain syringe, the bag of which should be held three or four feet above the patient's body. The child must lie on the back or left side, with legs well drawn up. The tip of the well-oiled catheter is passed into the rectum. When an introduction of two inches has been effected, allow the water to pass in slowly. The water will distend the parts and facilitate the further introduction of the tube. Press the folds of the buttocks together until the colon is filled. This in a child eighteen months of age will require twenty-four to thirty ounces of water. When this or a lesser amount, at least one pint, has passed in, allow the solution to run in and out at the same time.

The medical management of these cases of summer diarrhea among the tenement poor is important; but what is

equally important, and to which in a great measure our success is due, rests in the education of the mother. What these mothers fail in chiefly is cleanliness, and they are careless in caring for the food. They have no sense of appreciation of careful detail. It is manifestly desirable that the best milk within their means be given the child, and they are told where to obtain it; but an absolutely pure milk if kept uncooked, exposed in an open vessel in a very indifferent and dirty ice box at a temperature of 60° F. or over, would become contaminated at once.

In order to prevent diarrheal disease in summer the child should be fed properly all the year round. The death rate from this cause in June and early July is almost as large as in August; this is because the susceptible, the badly fed, those with chronic digestive disorders fall easy victims at the commencement of the heated term. The poor mothers are very much interested in their children, and when they make mistakes they are errors of ignorance, not of intent. If cow's milk is used they are told to boil it as soon as received, dilute it with a cereal water and keep it on ice.

They are also instructed in the care of the bottle and nipple. In case a special article of diet is to be given they are told how to prepare it; written directions are given covering these points. Nothing is left to the memory. The mother is told further that she must wash her hands with soap and hot water before touching the baby's food for any purpose, and that there must be a vessel half full of water into which the soiled napkins are placed until washed. With the first sign of intestinal derangement, whether in winter or summer, they are taught to stop milk at once, give a cereal water, a dose of castor oil, *and they do it.*

At the out-patient service we have many evidences of the benefits of teaching the mother. Many of them have been coming to us for years with the babies as they appear. Not infrequently the mother brings the child with a story of a sharp attack of diarrhea, a dose of castor oil, milk discontinued, barley water given, and the child on the road to recovery. Observing the good results of the suggestions, she goes about doing missionary work and tells her neighbors.

Our work, while covering a comparatively small field, shows that the large mortality of summer diarrheas in our large cities is preventable. The prevention rests with the people—the muni-

cipal government. If one-hundredth part of the expense and energy exerted by the rival political parties in New York City in a single campaign were devoted to the interests of the sick infant poor, how different would be my story. These infants cannot be moved to the country except in a ridiculously small number; they must live or die in their tenement homes. Hospitals cannot care for them, and but little advantage could be gained if this were possible. In summer diarrhea there must be isolation, not aggregation. I can treat with infinitely better success one hundred cases of summer diarrhea in the average tenement with the average well-meaning tenement mother than in the best equipped hospital with expert nursing and feeding. In the home the dangers of reinfection are slight. In the hospital, with the nurse going from patient to patient, she carries the infection from the virulent case to the susceptible, mild case and reinfects the convalescent.

The large summer mortality from intestinal disorders is not due to the tenement or directly to the hot weather, but to the absence of a little knowledge and the complete inadequacy of facilities which exists there during the hot months. In short there is a total inability to meet the changed conditions brought about by the hot weather. Dispensaries do excellent work in this respect, considering their inadequate appointments, cramped in space and a complete absence of trained paid assistants. If the recently graduated dispensary assistant were paid a small salary much better work could be commanded and obtained.

The municipality should establish milk laboratories and stations, one for a certain number of the poor population, where sterilized milk and cereal gruels and animal broths could be furnished free every month of the year to those who could not pay, and at a small cost to others. There should be at least one salaried physician and visiting nurse who could advise and teach mothers in the infant's care and feeding and furnish literature bearing upon the matter in the native language of the mother. An ice station should be connected with every laboratory and ice supplied free or at a small cost. If good food were provided for the well, and the mothers instructed what to do when the first sign of gastrointestinal disorders appear, the deaths from summer diarrhea would be reduced to a comparatively small number.

The above proposition at this time may sound utopian, but in the future when man in the process of evolution is still

further removed from his aboriginal ancestor who held life very cheap, his estimate of the value of this human life will have changed and there will be free milk laboratories and free ice stations, properly conducted by an unselfish municipal government supported by a humane people.

I am indebted to Dr. W. B. Hoag and Dr. John F. Connors for their valuable aid in carrying on the work at the out-patient department of the Babies' Hospital.

113 WEST EIGHTY-THIRD STREET.

DISCUSSION.

DR. CROZER GRIFFITH.—I have very few criticisms to make on this valuable paper, which expresses nearly my own views; and I have had an experience of thirteen or fourteen years of treating these diarrheal diseases in the out-patient department of the Children's Hospital, of Philadelphia. I remember a number of years ago making a series of experiments by treating these cases with different drugs and diets, selecting cases as nearly similar as possible, and giving one bismuth, another a tannic acid preparation, another opiates, and another only starvation for twenty-four hours. It was interesting to see that nothing was accomplished in any group unless the children were taken off of milk diet. It was also seen that it was absolutely necessary to use opiates in some cases. The feeling against opiates existing a few years ago was a mistaken one, and I believe we all recognize now that there are cases where the active peristalsis is the threatening factor, and that this must be stopped promptly. This does not, of course, for a moment mean that opium should be used in the majority of cases, for it is capable of doing great harm. So, too, in regard to irrigation, it is often over-done; and oftentimes I have seen a diarrhea cease after irrigation was stopped. I have entirely abandoned all astringent irrigating fluids, and now use nothing but salt solution or starch water.

I cannot agree, however, with the author in his condemnation of alcohol and of egg water. Each of these I regard as of the greatest service, and I would not like to be without them.

DR. KOPLIK.—I agree in the main with what the author of the paper has said, but I am surprised that he should abandon albumen water which I have found one of the most useful adjuncts during the first few days. I do not find that it is not digested. The principal trouble seems to me to be that the mother does not consider it nourishing enough. Last summer I made an attempt to use dextrinized gruels and found that they were not indicated until after vomiting had ceased. As to

opium, it is perhaps strange, but I very rarely use it, but by this I do not mean to infer that it should never be given.

The important point in regard to irrigation is that it should not be done too often and in the very severe cases I have rarely found it necessary to irrigate more than twice in the twenty-four hours. The point was made by the author that in the simple green movements irrigation was most useful and that in the severer cases he did not succeed in washing away very much. My object in resorting to irrigation is to cleanse the lower part of the bowel, of course, and also by using hot salt solution to stimulate the baby. In collapse irrigation is indicated. As for the public laboratories, we have them in New York, but they are not under proper control. In my dispensary we have the ideal system outlined by the author. The doctor sees the baby first, treats the acute illness and then sends the baby to another room at the proper time for the food according to its needs. Milk laboratories are a source of harm when not under the control of the physician.

DR. BUCKINGHAM.—There are a certain number of things in this paper that I should not agree with but I do not want to inflict my ideas of infant feeding on this Society. In irrigation the only good you are going to accomplish is the mechanical cleansing except where you desire absorption of salt solution as referred to by the previous speaker. It seems to me that as I have seen irrigation done a number of times it has been made unnecessarily rough by the use of a long tube, and I believe the results will be just as good if you use the ordinary syringe tip about an inch and a half long.

I do not think that milk laboratories should be under municipal control.

I cannot agree with the reader that brandy should never be given to infants with summer diarrhea and I would like to ask his reasons for that broad statement.

DR. HOLT.—There is just one other point that should have a little more stress upon it than has been given and that is that these children need rest, and I think it is a very important point that we should not order too many things to be done for them. I had a striking illustration a few weeks ago of how much harm can be done by persistent irrigation. I saw a baby in consultation, where two irrigations a day had been used for nearly seven months, first using salt solution and later tannic acid. This was done because of the persistent discharge of mucus from the colon. The pathological condition seemed clearly due to the treatment employed. The younger men have been taught that the presence of mucus in the colon in large quantities is an indication for irrigation and when the discharge continues they naturally think they have not done the washing with sufficient thoroughness. I believe thoroughly in

irrigation, but the time that it does most good is at the outset, when the intestine is filled with decomposing food and mucus. After thoroughly cleansing, irrigation should only be done to meet special indications.

I would like to add one more drug to the author's four and that is magnesia. Last year we treated in the Foundling Hospital a large series of cases with sulphate of magnesia as the initial cathartic, and it many cases it has certainly advantages over either calomel or castor oil. We gave ten or fifteen grains every half hour until clear watery stools appeared; generally it was necessary to give about two drachms.

DR. NORTHRUP.—If I had the chance to check off the many good points made by the author in his excellent paper I should check off the calomel followed by castor oil, for I have come to think that we should follow the calomel with castor oil, and I do not know of anything that cures so many patients for me as castor oil. I begin to think that if you should label it with some long name ending in "in" it would cure almost anything. Calomel plus castor oil, gum arabic water, starvation diet, rest and plenty of water are the points I would specially emphasize.

DR. CHAPIN.—I want to speak of one point in the technique of irrigation. I believe one trouble is found in the careless passing of the rectal tube. I have repeatedly seen the tube passed so that it simply bent on itself and a certain amount of water inserted which simply ballooned out the rectum and never passed the sigmoid flexure at all. In many cases I believe the best results may be secured by using a small tube, raising the child's buttocks and simply letting the water flow in slowly. Those who are not expert in the manipulation may do more harm than good in the attempt to introduce a tube into the child's bowel.

DR. FREEMAN.—I think there is one limit to be remembered in these cases as to the use of gruel. The weight of the children should be carefully watched and if they are losing rapidly and the discharges simply stain the diapers slightly, some more nourishing food should be given.

As to the administration of bismuth, a distinction should be made between its use in hospital and private practice, for it appears to do more good in private than in hospital patients. In the New York Foundling Hospital it has been used but apparently without doing any good. In private practice it is undoubtedly a very valuable drug.

DR. WINTERS.—I think we have had presented one of the most important and valuable papers that has been brought before this Society for many years. The discontinuance of milk at once should be recognized as of the greatest importance in these cases. As to the long discontinuance of milk and the

substitution of cereals I am in some doubt. Some gentleman has said that if cereals are not good for well babies how can they be good for sick ones. I went through this period of the use of various cereal preparations at the dispensary with the entire exclusion of everything else. I am not prepared to say that the results with cereals are entirely satisfactory either alone or in combination for any long continued service. I believe the milk should be stopped for at least twenty-four hours.

The drug treatment as laid down in this paper is perfect. I believe too that alcohol should never be used in these cases, if you must have a stimulant, very hot water is the best you can get. I give it so hot that it has to be used in drop doses and it should be given every fifteen minutes or so until these babies have taken as much as they can assimilate. I believe in the clearing out with calomel and oil. In some cases I give one first and in some the other. In regard to opium I find it a necessary adjunct in a limited number of cases. Irrigation has its place although it has been used to excess and a great deal of harm done thereby.

After having reached a period where you have quieted the local disturbance it seems to me that a weak milk solution, 1 or 2 per cent. of fat and .25 of proteids with 7 per cent. of sugar will do more good than cereals. Gradually it may be strengthened.

DR. ADAMS.—Unless the dispensaries are differently conducted in the large cities from what they are in the smaller ones the deductions must be different. I have been in the dispensary and hospital service, in Washington, for many years, and I have not been able to draw any such favorable deductions in diarrheal diseases from my service. It seems singular to me that in a city with a large foreign population where prejudice and superstitions are much greater than in the more American population of the smaller cities, that you can have your directions carried out so much better in the former than in the latter. I have very little confidence in the average dispensary patient carrying out directions after she passes from the observation of the physician. These people are so influenced by friends and environments that though their intentions be good you do not know what is being done after they pass from your supervision.

One of the greatest features of this paper is the effort to impress upon the general physician the importance of not putting anything into the gastroenteric tract that cannot be digested. Now that is a difficult proposition. The average man will not believe that you can keep a child alive without milk. He will ask, "What is it going to live on if you do not give it milk, that is its natural food and it must have it."

Dr. Holt struck the keynote about irrigation. Many children are benefited by it, but more are harmed, and I fre-

quently see cases in which harm has resulted. I indorse the method referred to by Dr. Chapin. The President saw a baby with me this year who was practically kept alive for ten days by normal salt solution administered in this way.

As far as bismuth is concerned I have used it a great deal in past years, and at autopsies I have seen the intestinal tract coated from the pyloric orifice to the anus, and yet the diarrheal process had kept up. I have almost ceased to use it and now depend upon stopping the milk promptly and letting the case alone to rest for a while. I do believe in the cereals and often get good results with them. As soon as we see that the stomach is able to digest albumen we should begin the administration of diluted milk.

DR. COTTON.—It seems to me that we ought to make the matter quite clear when we refer to rectal irrigation. Many physicians will read this discussion with some doubt as to its meaning. One authority recommends it in his text book and then expresses doubt as to its being used too freely. It seems to me it should be stated that rectal irrigation will serve different purposes under different conditions and that its use or discontinuance should depend upon certain cardinal indications. I do not believe there is any doubt in the mind of any member of this Society as to what he uses it for. It may be given at first to cleanse the colon and again to stimulate peristalsis of the small intestine to bring down into the colon the decomposing material and when that is accomplished we have done all that we can with rectal irrigation for a time. There is another rectal irrigation that means something entirely different. The moribund child with a weak heart needs irrigation in order to fill the vessels with fluid, and normal salt solution injected into the colon will stimulate such a heart. I believe of all these the most valuable is the first washing out of the large intestine.

As to feeding, I want to ask, what is the matter with egg? Is there anything about it that is contraindicated except the bad odor of the stools?

DR. MORSE.—I agree in almost every particular with the author's most admirable paper. I cannot agree with him as regards egg-albumen, however, as I have found it a very valuable substitute for milk in the first few days. As to the use of stimulants, I do not believe in them as a part of the direct treatment of summer diarrheas, but I do believe in them for the treatment of sick children. I feel certain that children sick with diarrhea often get into a condition where stimulation is necessary. regardless of the cause of the weakness. I also feel sure that it does no harm in these cases but much good.

DR. DORNING.—I would like to emphasize a point to which casual allusion has been made, namely the value of fresh air in the management of these cases of summer diarrhea. Before we

knew very much about intestinal antisepsis we used to send these cases to the seashore or to the mountains and many of them recovered without very much medication. I have myself repeatedly observed a marked improvement in the child's condition before it had gone more than twenty miles from the city on a river steamer.

Rest is an important factor in the treatment of these cases and that implies judicious, but not too frequent, irrigation of the bowel and not too much medication. Some caution should be observed in the use of beef juice. I have seen several cases suffer a severe relapse after the administration of beef juice. It is well to remember that tainted meat may give rise to severe ptomain poisoning. To avoid such a complication, when ordering meat juice, I have the outside portion of the meat cut away and use only the inner part in obtaining the juice.

Two cases have come under my observation in which symptoms of arsenical poisoning developed during the administration of large doses of bismuth subnitrate. The possibility of such an accident would suggest the advisability of endeavoring to obtain this drug pure when prescribing large doses.

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DR. ROTCH.—Will the author of the paper please tell us why he objects so strongly to the use of alcohol in these cases?

DR. KERLEY.—I do not use alcohol because of its tendency to produce or increase vomiting and because it is apt to be given in much larger quantities than was intended when prescribed. In many of these cases nephritis exists as a complication or sequela and I have thought that the tendency to nephritis was greater in the cases where alcohol had been used.

DR. ROTCH.—May I inquire why Dr. Kerley gives barley water in the first few days?

DR. KERLEY.—Because the patients keep it down and live on it.

DR. ROTCH.—There are many questions which ought to be settled before entering upon the discussion of summer diarrhea, and in the first place we should definitely agree as to what we mean by the term. In different parts of the country the term is used for different forms of intestinal disturbances, and I thought we had decided some years ago to drop the term when we adopted the new nomenclature and spoke definitely of cholera infantum, fermental diarrhea and ileocolitis. Whether those who use the term summer diarrhea mean it to describe an ileocolitis or something else, it is impossible to say, and hence when they speak of treatment it is difficult to say whether or not they are right. What I suppose they mean to discuss are the fermental diarrheas, since this class forms a large majority of the cases occurring in summer and presenting no organic lesion. If, on the contrary, it is ileocolitis they mean, then we are dealing with an organic disease which requires a differ-

ent form of treatment. Unless we say exactly what we mean by the term it is impossible to discuss the subject fairly.

There is no question but that in the out-patient department of the hospitals you cannot judge by numbers but by the thorough way in which the cases are examined. In an out-patient clinic you can only get through with a certain number of cases in a morning, and whether the deductions be made from one city or another makes no difference, we should judge from the individual cases. It is impossible to make proper deductions from the out-patient clinics as they are carried on at present with the feeding left in the hands of the mother at home. Until the feeding of the children can be placed on the same plane as the prescribing of drugs through the medium of the apothecary you cannot arrive at fair conclusions. This continually saying that you must make your deductions from your poor patients is a fallacy. You should start at the other end and finding out what is the best thing to do for a certain form of disease, carry it out as far as possible among the poor. To say that the mother will not give a drug and that therefore you do not order it does not make a profitable, general deduction. We have laboratories established in Boston and a fund for conducting them is placed in the hands of one man who supervises the conduct of all the dispensaries, and this fund is used for treating the diarrheal cases. In this way there is uniformity in the treatment and several hundred babies are under treatment at one time. We should thus try to treat the poor just exactly as we treat the rich and then we can make our deductions from the entire class.

Now it so happens that with this exact and systematized treatment my deductions are decidedly different from those made by some of the observers who have just spoken. In the first place, the author tells us to give barley water in these fermental diarrheas. Why? Because it is kept down better than anything else and is nourishing. It contains starch, and if you give much of that it will not be kept down. If the amount of starch given is small, the amount suggested, such as 1 per cent., it cannot be given with the idea of nourishment. Instead of giving starch in the form of barley water, why give anything at all. In the first twenty-four hours the digestive tract is not in a condition to absorb anything and the tissues, unless the case is one of cholera infantum, are not crying out for fluid at this time; they ought to be let alone. Simple, sterilized, distilled water in the first twenty-four hours is all that is necessary to allay the craving the infant has during the early hours of the disease. Why alcohol should not be given I cannot understand. These infants have nothing the matter with their kidneys during the first twenty-four hours of a diarrheal attack, and that is the time when a stimulant is often needed and it is also the time when you wish to tide the baby over this brief period until something can be absorbed in the way of nourishment. It may cause vomiting in

some cases, it does not in the majority. If the baby is in a state of collapse I should give it brandy even if it had something the matter with its kidneys.

I think this paper is a most admirable one for the purpose for which it was written. It covers the ground thoroughly and it is well to lay down just such rules as the author has done, but I do not believe his is the best way to treat fermental diarrheas.

In regard to the precision that can be obtained I think there are very few men who really appreciate how much can be done by knowing what are the constituents of the foods they are using. For instance, albumen water may do good but as a rule it is given in entirely too high a percentage. This difficulty can be obviated by ordering at the milk laboratories exactly the percentages of milk proteids which you require, for by some recently made observations at the Boston Laboratory it is now possible to have a fat free, a sugar free or a proteid free milk either alone or a combination of any one or two of these elements. Surely it is better to thus use milk put up in a clean, scientific and practical way with just the constituents we desire, than to continue to give albumen in the form of white of egg even though it may be, as is thought by some to be, a better form of albumen than that obtained from the proteids of cow's milk.

The giving of drugs, as a rule, is I think a great mistake. There is entirely too much calomel given in these diarrheal cases, and at times it is far from effective and often has to be supplemented by a saline. The treatment by irrigation is valuable in the beginning and at times during the course of the disease. Dr. Chapin's remark about the way to irrigate is very important and I think that the physician should at first do the irrigating himself and not leave it to the nurse.

DR. WINTERS.—I would like to ask the reader of the paper if he did not find it perfectly feasible to have his instructions to these mothers carried out absolutely.

DR. KERLEY.—Yes, I found it so.

DR. WINTERS.—That has been my experience. I have worked with these tenement house babies for many years and in my experience the mothers do absolutely as they are told and the instructions are carried out as accurately as they could be in the hospital. The results of the treatment of summer diarrhea, and I think it is a good term, at our dispensary are far more satisfactory than in the hospital. These babies cannot be congregated in any hospital without danger because summer diarrhea is a contagious disease. I believe that Dr. Rotch is right in regard to the nutritive value of barley water, but I do believe he is wrong in his description and nomenclature of summer diarrhea.

DR. ROTCH.—I would like to ask Dr. Winters his definition of the term summer diarrhea—what is the pathology of it?

DR. WINTERS.—I don't know that I exactly understand it, but I fail to comprehend the nomenclature that was recommended by this Society some years ago.

DR. ROTCH.—The nomenclature, instead of using the indefinite term summer diarrhea, mentions three distinct and well-known diseases: cholera infantum, fermental diarrhea and ileocolitis. Each has its own pathology, and each should be treated according to that pathology. It seems most extraordinary to me that the poor tenement people in New York can be so absolutely trusted to modify milk when they cannot anywhere else. They cannot be trusted to follow directions in Boston, Philadelphia or Chicago. I visited these people in one of New York's large out-patient clinics. I was much impressed with the belief that they could not be trusted to do fine work in modifying, and certainly not clean work. Nowhere in the world can ignorance and filth compete with intelligence and cleanliness, and yet this is what is upheld when you compare the work of the tenement house mothers with that of the milk laboratories.

DR. WINTERS.—These cases begin often as a digestive disturbance, and it is far better to describe them so than to refer to them as functional disturbances. There is no satisfactory classification at present to apply to them.

DR. ROTCH.—Do you mean that there is or is not a lesion?

DR. WINTERS.—In many cases I do not think you can determine at the time, and I do not believe the study of the pathologic lesion would aid us very much either in classification or treatment.

DR. ROTCH.—On the contrary it is this very study which we should continue as we would the pathological conditions in any disease. We must recognize that the summer diarrheas include a number of diseases differing pathologically and in their symptoms also, that this being the case the treatment differs decidedly. Even if we cannot always distinguish clinically one disease from another that is no reason why we should not acknowledge that this is due to our own ignorance and does not in any way prove that we should treat them all alike. In most cases cholera infantum can be distinguished from ileocolitis, and again either of these diseases from the fermental diarrheas; each of these diseases has its own definite treatment.

DR. WENTWORTH.—I should like to call attention to the lack of correlation of the symptoms to the pathological lesions in cases of diarrhea in infants. Dr. Booker's work has shown that in cases of summer diarrhea lesions are always present in

the intestines, although in many cases the intestines may appear to be normal macroscopically. Oftentimes very marked lesions are found in the intestines at autopsy in cases in which the clinical symptoms were scarcely noticeable. On the other hand very severe diarrheas may be associated with slight lesions in the intestines. The practical application of this is that a nomenclature applied to lesions in various parts of the intestinal tract in cases of summer diarrhea can only have a pathological interest because the diagnosis cannot be made clinically.

DR. SAUNDERS.—This exceedingly valuable paper speaks of what can be done in a large out-patient clinic. The classification will not be a guide to us either in private practice nor in the dispensary. As to the use of alcohol, the theoretical objection that it might produce or increase a nephritis should not deter us from using it. I have always used brandy or wine and I find the latter very useful and often accepted better than the former. I was surprised to hear distilled water recommended for I thought sterilized water was better for the system, because distilled water is a stranger to the tissues of the human body. The English writers mention the use of tea very frequently and I have found that it is an excellent stimulant and that the children become very fond of it. I would like to ask the author if he uses sugar in his cereal mixtures.

DR. KERLEY.—I do.

DR. SAUNDERS.—I have seen babies fed on cereals without sugar have a continuous subnormal temperature and I think the importance of giving sugar should be emphasized. The French writers are in favor of abstaining from any food whatever for the first twenty-four or thirty-six hours and I believe that in most cases that is the best treatment. As to the use of white of egg, I think we must consider the age of the patient, as babies under two or three months will not digest it. Two remedies that I consider important have not been mentioned, one is atropin which may be used to check the secretions in choleraic attacks and the other is chloral which is extremely valuable in securing rest for the patient.

DR. WILLIAMS.—There is one point which should be brought out in this connection concerning the difference in milk supplies. In Boston the milk is handled in such a manner that it is very old before it reaches the poorer people. I understand that is not the case in New York and it may explain some of the differences between the New York and Boston men.

DR. TOWNSEND.—I want to speak a word for the poor women of Boston. I have had excellent results in my work among them and have been able to get them to carry out my instructions.

DR. ROTCH.—Dr. Townsend will have still better results when he avails these poor women of the advantage of having

good fresh milk prepared at the laboratory through the summer milk fund. In regard to the objection to distilled water I think we have still a great deal to learn about it and why so many have spoken against its use in the past I do not know. I think I have been misunderstood. I should say that my experience has not only been in the hospital wards but with the poor people in their own homes, and that our summer milk fund especially provides for this class of diarrheas which having now for some years been treated by milk modifications from the milk laboratory enables us to judge regarding our results in the best and most exact way. On the other hand the deductions of those physicians who have treated their tenement house diarrheal cases with city milk modified by the mother's have no exact knowledge as to the percentages and the amount of dirt and bacteria which are influencing their results.

DR. KERLEY.—My article refers entirely to the 555 cases among the out-patient poor. If the patients had been differently situated perhaps we might have treated them differently, but we could not have had better results.

I do not give alcohol to these cases because it makes the child vomit or increases the tendency thereto; a great many develop nephritis, which is to my mind a marked contraindication to alcohol. Further, I look upon it as unwise to prescribe alcohol unless there is positive indication for it and then only when we cannot find a substitute which will do as well. Indiscriminate alcohol giving to children is widely practiced already and should not be encouraged by us. Nitroglycerin, strophanthus and other well known drugs answer as well when a heart stimulant is necessary.

In regard to the long continued use of cereal water, this is not done without making attempts at milk giving. The particular child referred to as not having taken milk for five months it was because milk did not agree and not because it was not tried repeatedly. This child was given scraped beef in connection with the cereal water, five or six teaspoonfuls a day. I have discontinued the use of the egg water chiefly because I find that I can get along better without it. The stomach in many of these cases is incapacitated for digesting it and it passes unchanged into the intestine and furnishes as favorable a putrefactive culture medium as milk. It is not the question of odor. Children fed on egg water require a longer time to get well, run a higher fever and altogether have a more serious illness than those in whom it is not used. Occasionally when a child tires of the cereal water a weak egg mixture may be given once or twice a day and may be taken care of.

Why give any food at all early in the attack? If I were to tell one of the out-patient mothers to go home and give the child no food, she may obey for a few hours and then she will

give milk or anything that she pleases; the child must be given something which the mother will look upon as nourishment. At first I begin with a very weak barley water mixture, one teaspoonful to the pint; this is increased as conditions allow. I agree with Dr. Dorning that beef juices and the broths have to be given cautiously. In some babies these substitutes have a decided laxative effect, which I referred to in the paper. They always should be given in small amount, one or two teaspoonfuls of beef juice to five or six ounces of barley water; one-half to one ounce of broth to five or six ounces of barley water. They serve to change the taste and add to the nutrition of the substitute. A few children will not tolerate the smallest quantity of broth or beef juice.

The term "summer diarrhea," I use advisedly, as I know of no better for the condition. Surprises at the autopsy on infants dying from summer diarrhea never cease, and those who have done even a moderate amount of this work will agree with me most heartily in this statement. No man can look upon a clinical picture of a case of this illness and state the nature and extent of the lesion in the gut. It is just as impossible to do this as it is for us to state the sex of an unborn child. What Dr. Rotch refers to is a pathological classification, and can only be made at the autopsy.

One of the speakers has not had particularly good success in handling the dispensary mother. With us the case is different. By careful attention to the case, by careful questioning and by recording the child's condition, by weighing and examining the child thoroughly we make it plain to the mother that we are interested in the child and thus gain her confidence so that she will follow our directions most implicitly. At too many out-patient services proper attention is not given to patients. They are treated in a careless and indifferent manner and we have indifferent and poor results. We do not allow the mothers to pass from under observation. If the child is too ill to be brought to the institution, some one of the staff of the out-patient service sees the child at its home and we teach the mother what to do and how to do it.

Bismuth has been given very severe tests and has not been found wanting. Not every case was treated with bismuth from the onset. Some were dieted and given no drugs at all; others were dieted and given calomel and castor oil for a time, and then various drugs, which have been advocated as useful, were brought into use. Among the large number of preparations used Squibb's subnitrate of bismuth, with or without the assistance of opium, was the only one that gave us satisfactory results.

The point that I particularly wished to emphasize in the paper was the necessity of cutting off milk with the first signs of illness, no matter how mild, and not resuming it until

the stools were fairly normal. The practitioner may select his own substitute, but should not give milk.

DR. ADAMS.—Do you mean to say that you supplement your dispensary work by following these patients to their homes?

DR. KERLEY.—Yes; in every severe case I either go myself or send an assistant.

CASE OF ARTERIOSCLEROSIS.

BY ALLEN BAINES, M.D., C.M.,

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The rarity of this condition in early life causes me to bring this case before the Society and to report it as fully as I possibly can get data. I can find only the cases published by Holt, which are but seven in number, and the report of Brill and Libman * which makes me feel that the addition of this one with specimens and slides will be of interest to this Society.

In none of the cases which I have read, can I find that the sclerotic condition existed so generally as in this, it being found that only certain vessels had undergone change whilst in my case, with the exception of the cerebral vessels, everywhere in the body the change was found to have taken place. During life in tracing the various superficial arteries, all gave the pipe stem touch and resistance which one generally associates with the calcareous condition of vessels frequently found in old age, and this will be seen to be verified from the specimens.

The etiology of the subject I do not purpose touching on, it is fairly well known and agreed upon, syphilis being probably the most frequent cause of its production amongst the young. Heredity, nephritis, scarlet fever, diphtheria, rheumatism, gout, alcoholic and lead poisoning being the chief factors other than syphilis.

Willie F., aged ten years and six months, born in Toronto, admitted to the Sick Children's Hospital, December 10, 1900.

FAMILY HISTORY.—Father living, healthy mechanic, always strong and healthy, says he was never ill, absolutely denies ever having had any specific disease.

Mother, living and healthy; no illness beyond child bearing; no miscarriages, nor any manifestation of syphilis. Brothers, three, living and healthy. Sisters, two, living and healthy. No signs of vessel change in any of them, nor any sign of hereditary syphilis or struma.

PAST HISTORY.—Had measles at seven years of age. For past two years had suffered from chorea, on and off; attacks

* *Journal of Experimental Medicine*, Vol. iv., Nos. 5 and 6.

lasting from two weeks to ten weeks. For several months he has suffered from nocturnal enuresis, always wetting his bed unless he got up to pass urine during the night. Drank water in large quantities, and if he should awake, would get up to take a couple of glasses of water. No history of scarlet fever nor diphtheria. Always strong and healthy, capable of outdoing most boys of his age at running and games requiring physical activity and endurance. Could get no history of snuffles or rash, his mother reporting him, with the exception of the chorea, as particularly healthy, never having had a suspicion of rheumatism.

PRESENT ILLNESS.—On the evening of December 3, 1900, he complained of severe headache. During the night had eight convulsions and vomited three or four times a watery fluid. The lymph nodes on the right side of neck became swollen. The family physician being sent for, ordered him to bed. On December 5th his mouth became filled with dark blood clots; an antiseptic mouth wash and stiptics were ordered. The bleeding being profuse, and not lessening, the doctor ordered his removal to the Sick Children's Hospital on December 10th.

EXAMINATION ON ADMISSION.—Patient pale and anemic, lips bluish-white, expression dull, languid and sleepy, fairly well nourished; body and head found in a filthy condition, necessitating several hot baths before being satisfactorily cleansed. Does not look intelligent, face and manner indicating low social status, eyelids puffy, breath extremely offensive, pervading the atmosphere of the whole ward. Teeth discolored and much decayed. Right second upper molar sharp and projecting horizontally into the cheek. The mouth full of blood clots which formed every hour or so; this blood appeared to proceed from a cavity in last lower molar and from ulcers on the cheek —four in number. Three of these were of the size of a ten-cent bit, one as large or larger than a twenty-five-cent piece. The edges were red, hard and raised, irregular in shape, covered with a yellow slough, which when touched bled freely. The gums blue-red, swollen and averted from the teeth, with a dirty yellow pus oozing between teeth and gum. Tongue tender and covered with a black hard coat. The filthy condition of the patient, the stench from the mouth and the large unhealthy ulcer suggested cancrum oris, but the rapid way in which the patient improved under the administration of chlorate of potash manifested the fact that the condition was stomatitis ulcerosa.

December 11th.—Mouth has been kept clean with antiseptic washes, but hemorrhage still continues. I ordered the bleeding points to be touched with solid perchlorid of iron and a small piece placed in the tooth cavity, which was bleeding freely. This application acted very well, the bleeding ceasing during the afternoon.

December 12th.—Condition of mouth much improved; no bleeding; tongue cleaning at tip; patient very drowsy; temperature, 100°; pulse, 110; vessels noticed to be generally hard and resistant to touch.

December 14th.—Marked improvement; urinalysis shows albumin 8 per cent., specific gravity 1009; microscopically nil; pain on deep pressure over right side of abdomen and back and over kidney; no edema; skin dry and harsh; gums still bleed on slight pressure. A small opening like a tiny white ring was seen on the gum, which probably was the open mouth of an artery, and from which most of the hemorrhage proceeded.

December 20th.—Had a severe epistaxis from right nostril, lasting for two hours, controlled finally by a saturated extract suprarenal capsule plug; ulcers rapidly healing; tongue clean and fetor of breath much improved.

December 25th.—Another severe attack of epistaxis, left nostril; application of suprarenal capsule extract, soon subdued.

December 27th.—Only one ulcer left on cheek.

December 28th.—A third attack of epistaxis. Careful examination of all palpable arteries revealed sclerotic condition; the blood current being cut off by pressure left them as hard, resistant cords, the walls being evidently sclerosed and the stream needing firm pressure to be cut off; heart enlarged, left border being to left of left nipple line. This condition of vessel wall readily explained difficulty of controlling the hemorrhages which had occurred from mouth and nose.

January 3d.—Epistaxis occurred; vessel could be plainly seen spurting from right septum.

Urinalysis shows albumin 3 per cent., specific gravity 1008; microscopic examination negative. Patient has been quiet and drowsy for the past three days; amount of urine passed in twenty-four hours, 29½ ounces.

January 7th.—Has great dyspnea; respiration rapid and labored. Precordial distress—hard cough with pain, especially over liver region; pulse 130, strong and regular; liver extends

three inches below costal margin, very tender on pressure; heart, apex displaced, downward and outward; impulse diffused but strong; a strong, rough, systolic mitral murmur; veins in neck prominent; alæ nasi, dilating.

January 8th.—Easier to-day; abdomen has become tympanitic; pulse, 125.

January 9th.—Bowels have not moved since January 6th, resisting all purgatives and enemata; abdomen still more tympanitic; pulse, 90; temperature, 95°; heart sounds weak.

January 10th.—Gradually weakened during the night; respirations very slow, intervals of five to twenty seconds; death at 10 A.M.

Treatment consisted of antiseptic mouth washes: soda sulphocarbolate solution every hour or two with local applications of solid perchlorid of iron and internally potassium chlorate and muriated tincture of iron; for dyspnea, morphin and strychnin were given, and towards the end glonoin and nitrite of amyl.

Prof. Anderson submits following microscopic report:

"KIDNEYS both show practically the same condition. The capsule shows marked fibroid thickening and beneath the capsule is an area in which there is great increase of the interstitial tissue so as almost to replace the parenchyma of the organ. In this area there is also an infiltration with large numbers of small round cells, mostly proliferated connective tissue corpuscles, but some polymorphonuclear leucocytes. Throughout the sections there is a well marked fibrosis, in some places almost replacing the tubules. Many of the glomeruli show extreme fibroid, hyaline fibroid or hyaline change. The vessels of the kidneys show all degrees of sclerosis, with narrowing of their lumina, some of the smaller ones being practically obliterated. Their walls in places present hyaline or hyaline fibroid changes. There is considerable polymorphonuclear infiltration of the interstitial tissue and glomeruli in all parts of the sections. The epithelium of the convoluted tubules especially is swollen, opaque and granular and often desquamating so as to fill up the tubules. Many of the tubules contain casts, hyaline, granular, or at times, partly hyaline and partly epithelial. In fact the specimens show the contents of the tubules in various stages of transition from the fairly definite epithelium to partly and completely hyaline material, giving a beautiful illustration of this

THE TREATMENT OF THE NASOPHARYNX IN SCARLATINA.

BY A. SEIBERT, M.D.,

New York.

One of the gravest dangers of scarlet fever patients consists in the invasion of the mucosa of the nasopharynx by those bacteria usually causing scarlatinous sore throat, namely by streptococci and their associates.

The infected tonsils may be washed over by an antiseptic solution if swallowed frequently (say every hour) in reclining posture, and thus a superficial disinfection of the visible pharynx may be accomplished. I have found the following mixture to be the most serviceable for this purpose:

℞ Tr. Iodi., 2, 0; Potass. Iodi., 1, 0; Aq. destill., 120, 0; Acid. Carbolic, gtts. x. *M. Sig.*—A teaspoonful every hour.

This may safely be given (by night and by day) for four or five days successively to children of one year of age as well as to adults. An experience of twenty years has failed to produce a single carbolic intoxication with this mixture. Now and then patients will complain of gastralgic pains after taking this solution for a day or two, due no doubt to artificial hyperacidity induced by the contact of this fluid with the stomach. Mild iodinism manifest by a slight watery discharge from the nose is not infrequently noticed, but usually disappears after a few days. As most patients with streptococcus-pharyngitis suffer from anorexia due to the suppression of the gastric functions and particularly the absence of hydrochloric acid in the stomach, it must seem but rational to induce the secretion of the gastric juice by well-known stomach disinfectants and astringents.

To clean and to disinfect the infiltrated mucosa in scarlatinous nasopharyngitis I have used irrigations with 1 to 5 per cent. warm solutions of ichthyol, repeated every six hours. A half pint is allowed to flow through the nares and the nasopharynx from a fountain syringe suspended about three feet above the patient.

These irrigations have given me excellent results during the last five years in all cases where the swelling of the parts permitted the fluid to flow into the nose and out of the mouth. But where the infiltration of the nasopharynx has so far advanced as to obstruct the passage-way between nose and throat, irrigations will be found insufficient, for the fluid then returns through the other nostril without coming in contact with the surface of this cavity. This obstruction usually begins to manifest itself about the fifth or sixth day of illness, and is most likely to develop there where insufficient or no local disinfection has been attempted.

To disinfect means to destroy the life of bacteria, to kill them. The authors of our modern American text-books do not advise effective disinfection of the scarlatinous sore throat. Holt (p. 1007, 1897) speaks of using "bland solutions for syringing" and considers "forcible swabbing a very doubtful expedient." Rotch (p. 557, 1895) states that "in young children it is often impossible to treat the throat locally" and relates the case of a two and a half year old child, "in which both sides of the neck were much swollen, but where the child refused to have any applications made to its nose or throat."

Bland solutions cannot destroy streptococci and staphylococci in the mucosa of the nasopharynx, at best they will flush away loose secretions. Leaving young children to their fate and to good nourishment in such an emergency because they refuse permission for energetic action, is a rather lame excuse for advocating comfortable, well-fed nihilism in handling such cases.

As before the introduction of Behring's serum therapy I had been an advocate of energetic local treatment of bacillary diphtheria, so I wish to state emphatically to-day that it appears to me imperative to locally destroy bacterial life in the scarlatinous sore throat whenever and wherever we can reach it. As soon as we have an effective serum therapy for scarlatina and its complications, we can discard local measures, but not a day sooner. The farther advanced the cases of scarlatinous nasopharyngitis are that we are called upon to treat, the more intense the swelling and the obstruction to deglutition and respiration, the more marked the involvement of the cervical lymph-nodes—the more energetic should be the attempts at local disinfection.

During the past year I have treated 6 cases of scarlatinous nasopharyngitis in which irrigations were insufficient in a man-

ner which readily overcame the obstruction. This consists in local applications with a 50 per cent. resorcin solution in alcohol. A report of the youngest case will best illustrate their value:

Female infant, nine months of age, breast-fed, strong, well-nourished baby. Had been ill for five days when I was called by the family to give antitoxin for diphtheria, in which their physician did not believe. I found a scarlatinous eruption (overlooked by the non-believer in antitoxin) over the whole body, a marked swelling of the cervical lymph nodes, a fetid, seropurulent discharge running from the nostrils, the head of the infant drawn far back to facilitate the difficult and audible respiration, and a grayish edematous infiltration of the pharynx, including the soft palate. The temperature in the rectum was 103.5° F.; stupor. The baby had not nursed for twenty-four hours. As an attempted irrigation did not bring the ichthyol solution into the nasopharynx, as the baby was septic and was evidently suffering from a streptococcus infection and not from bacillary diphtheria (proved later on by culture), I concluded that only energetic local measures could possibly save the infant's life. The patient was, therefore, placed upright on the mother's lap, while the father stood behind and held the baby's head, as in intubation. A cotton plug wound around the curved end of a wire was dipped into the resorcin-alcohol solution, gently pressed against the neck of the bottle to get rid of superfluous fluid (which might run into the larynx) and then introduced over the handle of a dessertspoon into one side of the nasopharynx. The soft palate instantly contracted and pressed the solution out of the cotton, thus spreading it over the inner surface of the nasopharynx in the course of a few seconds. The cotton was then withdrawn. Another fresh cotton plug was then introduced on the other side of the uvula and withdrawn instantly. *No force and no swabbing were employed.* One hour after these applications, which took but little more time to make than an ordinary throat inspection, and hardly gave more discomfort to the patient, the baby took the breast again, simply because the passage between the larynx and the nose had been opened up again. There was no hemorrhage. The next day I found the baby decidedly improved, for the swelling of the lymph nodes had subsided considerably, the temperature was lower and the baby could take the breast.

These applications were repeated once daily for five successive days. On the sixth day of treatment the throat was clean and the lymph node swelling had disappeared. Every application improved the condition of the patient visibly.

The other children treated in this manner were two and a half, three, four and a half, five and seven years of age. All recovered promptly from their nasopharyngitis; not one of them developed otitis. The second youngest (a boy two and a half years old) later on developed scarlatinal rheumatism, nephritis and empyema successively.

These resorcin-alcohol applications I have employed during the last eight years in the treatment of post-nasal catarrh in infants and children. My youngest patient was four months old. Contrary to Rotch's opinion, I have found it so that the younger the patient the easier it is to apply this treatment and the less opposition is made.

In closing I may be permitted to repeat what I said when advising inunctions of ichthyol-lanolin ointment for scarlatinous dermatitis and the irrigation of the upper air passages in scarlatinous sore throat by warm ichthyol solutions (ARCHIVES OF PEDIATRICS, 1895, and *Jahrbuch für Kinderheilkunde*, 1900), namely, that I consider it rational and imperative to attempt to destroy the pathogenic bacteria in scarlatina wherever they come within our reach (in the skin and in the throat) and to make these attempts as promptly and energetically as possible without injury to the patient.

These resorcin-alcohol applications have proved themselves to be perfectly harmless, and to my mind are indicated in scarlatina as soon as the nasopharynx becomes involved.

114 EAST FIFTY-SEVENTH STREET.

PERNICIOUS ANEMIA IN INFANTS, WITH A PRELIMINARY REPORT OF A CASE.

BY T. M. ROTCH, M.D.,

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AND MAYNARD LADD, M.D.,

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The present methods employed in the clinical examination of the blood have done much to differentiate the varieties of anemia in adults and to a lesser degree in infants. In spite, however, of the progress which has been made we are impressed by the insufficiency of the data concerning the severer forms of anemia in infants to be found in the literature of children's diseases, and the readiness with which statements are accepted on the evidence of comparatively few cases. The anemias of infancy have not been investigated so thoroughly as those of later life. The number of complete and systematic examinations in infants are so infrequently reported that we cannot help feeling that the subject deserves much more searching investigation than has yet been accorded it, and that a more extended study of the different anemias is of great importance to those whose interests are closely associated with the early periods of life.

Cabot, in his third edition of "Clinical Examinations of the Blood," in a short but admirable section on the "Anemias of Infancy," summarizes the present state of our knowledge in regard to the nature of the pathological conditions of the blood which occur in infants, and indicates clearly the difficulty of distinguishing from present data the different blood diseases in children. This difficulty is partly dependent upon the peculiarities and instability of the blood-making organs in infancy, but it is undoubtedly enhanced by the incompleteness of many

of the reported examinations of the blood and also by the comparatively small number of cases.

Fischl (*Jahr. f. Kinderheilk.*, 1899, p. 26) goes so far as to say that the blood examination in infants is of service only in determining the fact of the existence of a mild or severe anemia, but is of no value in the differentiation of the type of the anemia.

It hardly seems possible that the study of infants' blood has so far been so exhaustive and accurate in its details as to justify so extreme a position. We may hope that the future will, in infancy and childhood, as it has to a large degree in adults, give us a definite and rational classification of the different clinical types of anemia.

There are certain peculiarities in infants which may be reviewed with advantage, as they bear directly upon the difficulties which arise when we attempt to make a diagnosis from the examination of the blood.

Infants may in a low state of anemia have an enlarged spleen, which in many cases may be considered as secondary to some primary disease, such as syphilis, rachitis and chronic malaria. On the other hand, there may be marked enlargement of the spleen, without any demonstrable antecedent cause, with or without pronounced leucocytosis, and the blood may not show the proportion of white blood corpuscles characteristic of leucemia. Cases of the latter class have been designated by von Jaksch as *anemia infantum pseudoleucemia.* In these cases the red blood corpuscles will often show changes which may closely simulate the blood of pernicious anemia very much in the same way that, in the leucemias of adults, the red cells may assume all the characteristics of progressive pernicious anemia. This class constitutes the greater majority of the very severe anemias of infancy, and in the literature is sometimes classed as pernicious anemia, and sometimes as leucemia, or, again following the lead of von Jaksch, is put into a separate class by itself.

It is not the purpose of this paper to enter deeply into a discussion of this type of anemia or pseudoleucemia. It represents a class intermediate between pernicious anemia and leucemia. In its typical form, showing pronounced leucocytosis with marked enlargement of the spleen, and often of the lymph nodes of the neck, axillæ and groin, it represents a very different

disease from the idiopathic pernicious anemia of infancy, characterized, as a rule, by only a slight increase in the white blood corpuscles, by little if any enlargement of the spleen and lymph nodes and by a blood which in respect to the red blood corpuscles is identical to that which occurs in the pernicious anemia of adults. We shall, therefore, exclude from further consideration this large and interesting class of anemia infantum pseudoleucemia, as the case which subsequently will be presented represents in a very striking manner the clinical symptoms and blood appearances of an idiopathic pernicious anemia, occurring in an infant nine months old.

Authorities are not agreed as to the use of the term primary anemia, some maintaining that all anemias are secondary or symptomatic. This claim is probably justifiable theoretically, but the examination of the blood by modern methods enables us to separate certain forms which, with a few exceptions which will be mentioned later, represent a severe and usually progressive affection of the blood, ending fatally in the great majority of cases and for which no cause can be found even after the employment of all the resources of the most advanced clinical investigation.

These cases of primary pernicious anemia are of rare occurrence in childhood and are still more rare in infancy, that is, in the first two years of life. They have been especially studied by Monti and Berggrün, d'Espine and Picot, and cases have also been reported by Retschlag, Huguen, Ellben, Baginsky, Variot and Mott. Audeoud, up to 1897, was able to collect only 25 cases, exclusive of cases due to intestinal parasites and poisoning by carbon monoxid, occurring prior to fifteen years of age. Nine of these occurred between the ages of ten and fifteen years; 8 between six and ten years; 4 between three and five years; and only 4 occurred in the period of infancy, the earliest being reported at three months. Death occurred in all of Audeoud's series.

In 2068 cases of infants under two years of age who were admitted to the wards of the Infants' Hospital, Boston, from January 1, 1882, to January 1, 1901, there has not been a single case of true idiopathic pernicious anemia. In the out-patient clinic of the same hospital there have been treated in fourteen years 72,644 cases; if there was any case of idiopathic pernicious anemia in this large number of infants and children, the

condition was never recognized, judging from the catalogue of diagnosis.

The youngest case of pernicious anemia on record has been reported by Demme (*28 Medic. Jahresberichte uber die Thatigkeit des Jenner'schen Kindes-spitales*), occurring in a breast-fed infant three months old. The red cells were said to have been 800,000 and the hemoglobin 28-30 per cent. The child died. We have not been able to obtain the original report, and do not know about the other important characteristics of the blood.

A second case (cited by Osler, *Twentieth Century Practice of Medicine*) was reported by Monti and Berggrün in an infant six and one-half months old.

Our own case at nine months would seem to be the third youngest.

A fourth case in an infant eighteen months old has been reported by W. Steffen, Jr. (*Jahr. f. Kinderheilk.*, Bd. XXVIII., p. 444). From the history, fragmentary blood examination and autopsy, the case was in all probability a case of pernicious anemia, but the examination of the blood is unfortunately not as detailed as one would like.

Two other cases, one in a child of eleven months and one in a girl of two years, have been reported by d'Espine and Picot (*Revue de Medicin*, 1890, p. 859), but it is difficult to accept the diagnosis in the case of the two years old child in the absence of both blood counts and autopsies. There was no blood count in the eleven months old infant, and the autopsy, while suggestive, did not give conclusive evidence.

A seventh case has been reported by Baginsky in an infant of two years (*Traite Mal. Enf.*, 1899).

After the age of two years the reported cases, while infrequent, are not so rare as was formerly supposed and are, in many cases, better attested.

All of these cases occurring during the period of infancy have died with the exception of our own, which will be spoken of later.

ETIOLOGY.—The changes in the blood which have been considered characteristic of pernicious anemia have been shown to occur in certain definite conditions. Arslan has reported what might almost be called an epidemic of severe anemia with the blood characteristics of pernicious anemia occurring in 21 infants

and young children, in whom the cause was due to an intestinal parasite, the ankylostoma duodenalis (Arslan, "Anemie des mineurs (ankylostome) chez les enfants," *Revue Mal. Enf.*, 1892, p. 555). Many other similar cases are recorded. The bothriocephalus latus and the ascaris lumbricoides have also produced this form of anemia. Koren (*Arch. f. Kinderheil.*, T. XV., p. 390) has reported three cases of pernicious anemia due to carbon monoxid poisoning followed by death in one of them, a boy of eight years. Syphilis, as shown by the cases of Laache, Kjernes and Muller, chronic malaria, as shown by the studies on the bone marrow of fatal cases by Bignami and by Dionisi, and myeloid sarcoma, in one case described by Litten, have all been advanced as direct etiological causes of pernicious anemia. The present tendency is, however, to exclude from the category of progressive pernicious anemia all those cases in which the etiology is known and to retain the term for those cases of severe anemia, with the characteristic changes in the blood, in which no sufficient primary cause can be proved.

Many theories as to the pathogenesis of idiopathic pernicious anemia have been advanced, but none have found general acceptance. Ponfiks has attempted to attribute the cause to a fatty degeneration of the heart, and many other writers have considered the disease due to an atrophic condition of the glands of the alimentary tract. There is, however, quite as much evidence that these pathological lesions are secondary and not primary, and this assumption seems much more likely when we consider how greatly out of proportion is the severity of the anemia to the changes in the different organs of the body.

Brigidi and Sasaki have tried to show a causal relation between the sympathetic nervous system of the abdomen and pernicious anemia, but although such lesions often occur they are not so constant as to be considered a primary cause of the disease.

Baginsky, Aufrecht, Frankenhause, Henrot, Bernheim and Petroni have been led by the frequency of fever and by the slight hyperplasia which is sometimes seen in the spleen to seek for some specific microbe, but no positive results have been obtained.

Hayem considers pernicious anemia to be due to a disease of the blood-making organs, by which the reparatory function

becomes disturbed or arrested, and the severity of the anemia to be proportionate to the degree to which the interference with the blood manufacturing organs is carried. Cabot makes the pertinent observation that "in view of our ignorance of the blood manufacturing functions, there is little difference between saying that a primary anemia is a disease of the blood-making organs and in saying that it is one whose cause is unknown, especially as the pathological appearance in the bone marrow, recorded in cases of so-called primary anemia, do not differ from those which can be brought about by bleeding. There is no good evidence that there is any primary disease of the blood-making functions." It is conceivable that in the future definite causes for all of these idiopathic pernicious anemias may be found.

Hunter has in explanation of the pathogenesis of pernicious anemia advanced the theory of an autointoxication arising in the alimentary tract and thence entering the general circulation by absorption. In a study of some of his cases he found in the urine an excess of aromatic sulphates derived from the sulphur of the proteid material of the intestinal tract, indicating a pathological increase in the putrefactive processes. In still further support of this theory of autointoxication he isolated from the urine three special ptomains which he thought might be the specific poisons. But here again we are met with the lack of evidence that these ptomains are the primary cause, for they may rationally be looked upon as the secondary conditions resulting from an abnormal fermentation due to the digestive disturbances which are so frequently present in pernicious anemia. The studies of Arslan rather support this objection to the primary autointoxication theory. In his twenty-one cases of pernicious anemia of parasitic origin in infants, he succeeded in extracting a toxin from the urine, inoculated it in increasing doses into rabbits and produced the characteristic symptoms of pernicious anemia. When the inoculations were stopped, the rabbits gradually returned to their normal condition, very much in the same manner that a child with symptoms of pernicious anemia, due to the ankylostoma duodenalis, will recover as soon as the parasites are expelled. In one experiment it was found that the urine from the children from whom the worms had been expelled did not exercise any toxic effect on the rabbits.

Koren's three cases of carbon monoxid poisoning, in which the symptoms and blood were typical of pernicious anemia, add weight to the intoxication theory, but as yet we have no evidence as to the nature of the poison nor as to the manner in which it is produced.

Audeoud summarizes and combines these various theories somewhat as follows: The infant, like the adult, can be exposed to certain intoxications from within, such as may arise from indigestion, nephritis, infectious diseases, etc., or from without, as in the case of carbon monoxid poisoning. These may produce pronounced alterations in the blood and other tissues. The organism combats the disease by putting into superactivity the blood-making organs. The production of these organs is limited; if the organism is feeble and the cause continues to act, the bone marrow, spleen and lymph nodes are not capable of their task and as the renovation of the blood is incomplete, the disease terminates fatally. If, on the contrary, the individual is young and vigorous, and the blood-making organs can make sufficient repairs, and above all, if the primary cause ceases to be active, recovery may take place. This might explain the lesser frequency of pernicious anemia in infants in whom the blood-making organs regain their activity with greater ease than in adults.

PATHOLOGY.—The pathological changes in the pernicious anemia of infants, so far as we can judge from the limited number of autopsies, show no essential differences from those which occur in adults. There is excessive pallor of all the tissues of the body, but rarely emaciation. It is not infrequent to find punctate or extensive hemorrhages into the different organs. In Cabot's series of 110 cases, hemorrhages were noted in 37 (*American Journal Medical Sciences*, August, 1900). The most typical lesions are in the bone marrow which is red and in many respects embryonic in type, containing large numbers of nucleated red blood corpuscles, especially the larger variety of megaloblasts. Fatty degeneration of the heart, liver, pancreas, kidneys, and of the muscular coats of the intestines is generally pronounced. The stomach and intestines show, as a rule, a well-marked glandular atrophy with or without interstitial proliferation and often with hemorrhagic extravasations or edematous infiltration. The spleen generally shows no important changes. Its enlargement could be demonstrated in only 13 of

Cabot's series, while the liver was enlarged in 30 cases. Eichorst, Quincke and others attribute this enlargement of the spleen to the degree of fever present during life rather than to a special activity of the spleen in the destruction of the red blood corpuscles. The liver is usually fatty and contains large deposit of iron in the outer and middle zones of the lobules. This deposit of pigment is considered by Hunter as a special characteristic lesion peculiar to pernicious anemia. It is also found in excess in the spleen and kidney.

The mesenteric lymph nodes are often hyperemic and may be increased in volume. Changes in the ganglion cells of the sympathetic nervous system have been reported on several occasions, but not in children so far as we know. Retinal hemorrhages were present in 15 out of 36 cases examined in Cabot's series.

The most important lesions from a clinical point of view are found in the blood. This, when freshly drawn, shows very feeble powers of coagulation and does not form in rouleaux. According to some observers ameboid motions of the poikilocytes and occasionally active locomotion of the microcytes may be seen. The blood is reduced in bulk and the specific gravity may run as low as 1027. The number of red blood corpuscles is greatly diminished, averaging between 1,000,000 and 2,000,000. Demme's count of 800,000 in an infant of three months appears to be the lowest in the pernicious anemia of infancy. Quincke's case, occurring in later life with 143,000 red cells, is the lowest count on record. The hemoglobin is absolutely much diminished and may be as low as 10 or 20 per cent. A characteristic feature, however, is the relatively high percentage of hemoglobin in the individual red cells, the color index often being increased. During the periods of improvement or remission of the disease, however, the color index may be normal or even diminished. The total number of leucocytes is generally below the normal, but this rule does not hold in infants in whom any anemia may cause an increase in the white count. The percentage of lymphocytes is usually increased at the expense of the polymorphonuclear leucocytes. The presence of a few myelocytes is not uncommon. Of equal importance with the extreme reduction in the red cells is the presence of nucleated red corpuscles, especially the large variety known as megaloblasts which outnumber the normoblasts, also of large non-

nucleated red corpuscles or macrocytes and of deeply staining red cells, or polychromatophiles. The average diameter of the red cells is usually increased and serves as an important aid in the diagnosis. Often large numbers of oval and pyriform cells are seen, and are considered very characteristic of pernicious anemia. Poikilocytes in large numbers and microcytes, in small numbers, as compared with the macrocytes, are present.

SYMPTOMS.—The symptoms of pernicious anemia in infants may be shown to the greatest advantage in the case which has come under our observation, and which we present to you in a preliminary report, as time alone can make its history complete.

J. T., a girl, was born on December 1, 1899. She has been under our observation from the day of her birth until the present time. There is one other child four years of age, and in perfect health. The family history is negative, in so far as this particular case is concerned. The labor was easy and the infant at birth was in all respects of normal physical development. She weighed seven and one-half pounds.

For the first few hours she was given a very weak modified milk, and was then fed by the breast entirely. During the first three or four days of the colostrum period she lost six ounces, at the end of two weeks she was gaining at the rate of an ounce a day, but showed the symptoms of a slight intestinal indigestion. At the end of three weeks the digestion was normal and the baby's weight had increased to eight pounds seven ounces.

At this time the mother developed a mild case of mastitis, causing some disturbance in the milk, in consequence of which the infant again showed symptoms of intestinal indigestion which, though not severe, were still persistent. In spite of the condition of the bowels the gain in weight was steady and at the end of sixteen weeks of breast-feeding, she weighed thirteen pounds and seven ounces.

When about four months old she began to lose in weight and the symptoms of intestinal indigestion became marked. The mother had plenty of milk, but it was found impossible to regulate its quality, and ten days later it was found necessary to adopt a substitute food. The child was put on a laboratory mixture beginning with a 3.50 fat, 7 sugar and 1 proteid, which was soon changed to a 4 fat, 7 sugar and 1 proteid. She then throve and appeared much better than for some weeks

previous to the change from the breast-feeding. She gained one pound in ten days. She was in every respect a fine specimen of an infant.

When about five months old she had a mild attack of measles, from which the recovery seemed complete. She continued to grow and appeared in perfect health so that when about six months old her weight was sixteen and one-quarter pounds.

During the latter part of June and during July and August she had some indigestion which required, from time to time, changes in the quality of the milk. She was then living on the North Shore and with every possible provision as to feeding, hygiene and nursing. She was out of doors much of the time and became very much tanned. She gained in weight and strength, was unusually large and robust and seemed perfectly well, with the exception of the temporary mild attacks of indigestion. During the latter part of August, however, she began to lose in strength and a slight pallor was noticeable. She was then eight months old.

On returning to town in October an improvement appeared to take place, her digestion became normal and the percentages in the milk were gradually increased to fat 4, sugar 7 and proteids 2.

It is impossible to state when the symptoms of pernicious anemia began. If the occasional indigestion in the months of July and August had any bearing on the subsequent disease, there was nothing in the child's physical condition to indicate any serious trouble, and repeated physical examinations failed to excite any suspicion of a serious underlying disease.

During the latter part of September, however, when the infant was a little over nine months of age, she was noticed to fail, not at all rapidly, but on the contrary in a very insidious manner. Indigestion again set in. The child began to have a raised temperature and a slight coryza. There was some fretfulness and restlessness, which could easily have been explained by a disturbance due to dentition. The indigestion ceased for two weeks. Then came a period of malaise and loss of strength. The child grew pale, the pallor becoming more and more noticeable as the tan of the summer wore off.

On November 4th, when ten months old, the pallor had become quite pronounced, and the hands and feet were noticed

to be slightly puffed. The temperature was 100° C. both morning and evening.

On November 5th, a physical examination was again made. Pronounced pallor, general weakness and a faint systolic murmur heard all over the precordia and in the neck, without any cardiac enlargement, and considerable puffiness of the face, legs, ankles, feet and hands were the only signs of importance. The murmur had not been noticed before. There were no evidences of rachitis or scorbutus, and no subcutaneous hemorrhages. There was no glandular enlargement to be detected, the spleen not being palpable and giving the normal percussion limits.

An examination of the urine showed it to be high in color, acid, clear, and with a very slight amount of flocculent sediment; specific gravity, 1012; albumin, slightest possible trace, giving with the nitric acid test a distinct zone due to uric acid; chlorids, slightly diminished; sugar and bile pigments were both absent; urea, 1.01 per cent.; the total twenty-four hour amount of the urine could not be obtained.

Microscopically the sediment consisted of uric acid crystals in clumps, associated with a few round mononuclear cells from the neck of the bladder and a few squamous cells free and clumped. There was an occasional leucocyte, but no blood nor casts were seen.

There was evidently nothing more than a slight passive hyperemia of the kidney with a very mild irritation of the bladder. The edema could not be accounted for by the condition of the kidneys.

The blood examination on the same day gave the following results: November 5, Hemoglobin, 30 per cent.; red blood corpuscles, 1,732,000; white blood corpuscles, 9,200. A differential count of 500 cells showed: Polymorphonuclear neutrophiles, 32 per cent.; lymphocytes, large and small (small lymphocytes in excess of the large round cells), 66.2 per cent.; eosinophiles, 1.8 per cent.

During the count 3 megaloblasts and 3 normoblasts were seen. There were numerous poikilocytes and cells of pyriform and oval shape. There were many large red corpuscles or macrocytes. The average diameter of the red blood corpuscles was distinctly increased as compared with the normal, although there were some abnormally small cells, or microcytes. The

color index of the individual cells was difficult to judge owing to the fact that the spreads were somewhat overheated.

The examination of the blood showed beyond question the presence of either a very severe secondary anemia or of a primary or pernicious anemia. The blood by itself was strongly suggestive of the pernicious type.

Every effort was made to determine a cause for a secondary anemia. The urine and several specimens of wall paper and carpets were examined for arsenic with negative results. Every possible source of poisoning, such as the cooking utensils, was investigated, but no good evidence of any could be obtained. The fecal discharges were inspected daily, and there was no suggestion of intestinal parasites. The attacks of indigestion had never been severe, and the splendid nutrition of the child for nine months of its life on a food, first of breast-milk for four months and then a carefully supervised laboratory milk, eliminated improper food as a cause of anemia. It was, therefore, impossible for us to discover any rational cause for so extreme a degree of anemia and weakness.

The infant failed steadily; the loss in strength, the increasing edema and the intensification of the hemic murmur being the principal signs of importance. The temperature varied between 99.5° and 100°. The food was well digested. There was some loss of weight, but no emaciation. There was no vomiting and no diarrhea.

The treatment consisted of iron, brandy, and careful nursing and feeding.

Several later examinations of the urine showed essentially the same analysis as reported above, but the urine became more diluted and the uric acid crystals disappeared.

On November 10th, Dr. R. H. Fitz was called in consultation, and was then inclined to consider the case as one of secondary anemia of unknown cause, although admitting that the character of the blood was presumptive evidence of pernicious anemia.

Although there was no evidence which suggested that the milk from the laboratory was in any way responsible for the severe and progressive condition of anemia and loss of strength, milk was obtained from another herd of cows, and cereals and broths were tried, but as both of the latter caused symptoms of gastric indigestion they were discontinued.

For two weeks the temperature (rectal) ranged from 99.6° to 101°, and then became higher and more irregular, on several occasions reaching 103° F. The pulse became more rapid and at times was irregular.

On November 20th, there was a slight collapse, the pulse suddenly rising to 200 and becoming very weak, and the respirations to between 50 and 60 per minute. The child was sleepy, weak and languid. The swelling of the feet, hands and eyes were more pronounced. The loss of weight in one week amounted to 1¼ lbs. The pallor was at this time extreme. The heart's action was irregular, and the systolic murmur loud and transmitted in all directions. The physical examination revealed nothing new. There was no chill, cyanosis, nor evidence of hemorrhage from any source.

On November 21st, another complete examination of the blood was made, which showed a rapid deterioration of the blood in the three weeks which had elapsed from the first examination. It was as follows: Hemoglobin, 20 per cent.; white blood corpuscles, 18,800; red blood corpuscles, 1,088,000; Widal reaction, negative; examination for plasmodia, negative.

The color index of the individual red blood corpuscles in both the fresh and stained preparations was nearly normal, and was relatively high as compared with the great diminution in the red blood corpuscles.

Differential count, 500 cells: polymorphonuclear neutrophiles, 43.4 per cent.; lymphocytes (small lymphocytes being in excess of the large and transitional forms), 54.2 per cent.; eosinophiles, 0.6 per cent.; myelocytes, 1.8 per cent.

During this count there were seen 9 megaloblasts and 3 normoblasts. The average diameter of the red cells was much increased. There was considerable poikilocytosis. The presence of very large cells or macrocytes was marked, and many of the cells were oval and pyriform in shape. There was moderate polychromatophilia.

The character of the blood was by this examination shown beyond any question to be typical of the blood of pernicious anemia in adults, in all respects except for the presence of a low white count. The tendency to an increase in the white cells in almost any severe pathological process in infants is so well-known, however, that no special significance can be attached to this variation from the adult type of blood in per-

nicious anemia. Moreover, the case fulfilled all the other requirements of pernicious anemia. A most searching investigation failed to determine any cause to which the anemia might be secondary. The absence of any enlargement of the spleen and the comparatively low white count in the majority of cases was sufficient to exclude an anemia infantum pseudoleucemia. The onset was insidious, the pallor extreme, the edema of the face, hands and legs was marked; and the languor and physical weakness rapidly increased. The temperature varied between 102° to 103° F., and the pulse averaged about 160. The infant was at this time so weak that she could not raise her legs or head and could scarcely move her arms. The general condition was such that the gravest fears were entertained regarding the result. At this time, November 21st, the inhalation of oxygen was tried, the oxygen being given for ten minutes every twenty minutes day and night whether the infant was asleep or awake. Coincidently with this treatment a marked change took place in the condition of the child as was seen by the gradual defervescence of the temperature, by the steady improvement in the rate and quality of the pulse and by the perfectly evident improvement in the color and strength. The food, which was of the same quality as in the previous three weeks, was taken with more vigor, the apathy became less marked, and the infant took more notice of what was going on about her.

Two days after this sudden change for the better, Dr. Abraham Jacobi, of New York, saw the child in consultation. He agreed unequivocally in the diagnosis of pernicious anemia, both from the clinical history and from the result of the blood examination. At his suggestion a weak solution of liquor potassii arsenitis was given and small doses of ferratin were substituted for the saccharated carbonate of iron which had up to this time been taken. The oxygen, which was continued with twenty minute intervals for two weeks, was then reduced to one hour intervals and then gradually diminished until at the end of six weeks it was entirely omitted. At the end of a week the arsenic was discontinued, as there were signs of its physiological action, and although it was tried again the infant was unable to tolerate it.

The subsequent history of the case was one of steady improvement, interrupted only by a slight serous otitis media, a

few attacks of coryza and one of influenza. The color improved, the strength returned, the systolic murmur and the edema disappeared and the infant began to gain in weight. Its maximum weight during the summer was twenty-five pounds; at the height of its disease, *i.e.*, on November 21st, it was about nineteen pounds, and in the past six months it has regained the six pounds it had lost. To-day, May 18, 1901, the infant is eighteen months old and weighs twenty pounds. It is apparently in perfect health, and its blood so far as can be determined, is that of a normal infant.

Examinations of the blood were made at regular intervals and it is interesting and instructive to notice the changes which have taken place since the lowest point was reached on November 21st.

On December 5th, two weeks after the beginning of the inhalations of oxygen, the blood showed the following characteristics: Hemoglobin, 35 per cent., a gain of 15 per cent. in two weeks; white blood corpuscles, 10,800; red blood corpuscles, 2,324,000, a gain of 1,136,000 in two weeks.

The differential count of 500 cells was: Polymorphonuclear neutrophiles, 75 per cent.; lymphocytes (the small lymphocytes being in excess of the large and transitional forms), 21.5 per cent.; eosinophiles, 3 per cent.; myelocytes, .5 per cent.

During the count there were seen: Megaloblasts, 3; normoblasts, 2. The average diameter of the red cells was slightly increased. Deformities were slight. There was no polychromatophilia. The hemoglobin per corpuscle normal.

Dr. Richard C. Cabot examined the stained specimens of November 21st and December 6th, and returned the following report:

"Both sets show characteristics which in the adult would point conclusively to pernicious anemia, and while I do not feel ready to speak so positively of a baby's blood in view of the limited knowledge of babies' blood which we possess, I yet believe that the case is, in all probability, *pernicious*, and that the baby will die within a year."

On December 19th, two weeks after the third examination and one month from the height of the disease, the examination of the blood gave the following results: Hemoglobin, 60 per cent., a total gain of 40 per cent.; white blood corpuscles, 15,200; red blood corpuscles, 3,692,000, a total gain of 2,604,000, or a gain in two weeks of 1,368,000.

The differential count of 500 cells gave: Polymorphonuclear neutrophiles, 59 per cent.; lymphocytes (the large and transitional form being in excess of the small lymphocytes), 34 per cent.; eosinophiles, 6.6 per cent.; myelocytes, .4 per cent.

During the count and in a careful subsequent examination of several specimens, no megaloblasts and but one normoblast was seen. The increase in the average diameter of the red cells was still apparent, although less marked than before. Poikilocytes were still present.

On January 2, 1901, two weeks later, six weeks from the height of the disease, the examination of the blood was as follows: hemoglobin, 70 per cent., a total gain of 50 per cent., or a gain in two weeks of 10 per cent.; white blood corpuscles, 11,000; red blood corpuscles, 4,216,000, a total gain of 3,128,000, or a gain in two weeks of 524,000.

The differential count of 500 cells gave: Polymorphonuclear neutrophiles, 60 per cent.; lymphocytes (the large and transitional forms being in excess of the small lymphocytes, 35 per cent.; eosinophiles, 4.8 per cent.; eosinophilic myelocytes, .2 per cent. During this count and on subsequent search of the stained specimens, no nucleated red cells were seen. The average diameter of the red cells was still slightly increased and a small number of poikilocytes still persisted.

The oxygen was omitted at this time, January 2, 1901.

On January 16th, two weeks later, and eight weeks from the height of the disease, the examination of the blood was as follows: Hemoglobin, 70 per cent., a total gain of 50 per cent., but no gain from the previous count; white blood corpuscles, 11,800; red blood corpuscles, 4,560,000, a total gain of 3,472,000, or a gain in two weeks of 344,000.

The differential count of 500 cells gave: Polymorphonuclear neutrophiles, 58.8 per cent.; lymphocytes (the large and transitional forms being in excess of the small lymphocytes), 35.2 per cent.; eosinophiles, 6 per cent. No nucleated cells were seen and the blood was essentially the same in other respects as on January 2d.

On January 30th, ten weeks from the height of the disease, the examination of the blood was as follows: Hemoglobin, 75 per cent., a total gain of 55 per cent., or a gain in two weeks of 5 per cent.; white blood corpuscles, 11,800; red blood cor-

puscles, 4,884,000, a total gain of 3,876,000 cells, or a gain in two weeks of 668,000.

The differential count of 500 cells gave: Polymorphonuclear neutrophiles, 52 per cent.; lymphocytes (the large and transitional forms being in excess of the small lymphocytes), 41 per cent.; eosinophiles, 6.4 per cent.; mastzellen, 6 per cent. No nucleated red cells were seen.

On February 20th, thirteen weeks after the height of the disease, the examination of the blood showed: Hemoglobin, 75 per cent., a total gain of 55 per cent., but no gain over the previous examination; white blood corpuscles, 10,500; red blood corpuscles, 5,292,000, a total gain of 4,204,000, or a gain in three weeks of 408,000.

The differential count of 500 cells gave: Polymorphonuclear neutrophiles, 75.6 per cent.; lymphocytes (large and transitional forms slightly in excess of the small lymphocytes), 14 per cent.; eosinophiles, 1.4 per cent. No nucleated red cells were seen and the blood was, to all appearances, practically normal.

On March 12th, seventeen weeks after the height of the disease, the blood showed: Hemoglobin, 80 per cent., a total gain of 60 per cent., or a gain of 5 per cent. in four weeks; white blood corpuscles, 10,600; red blood corpuscles, 5,372,000, a total gain of 4,284,000, or a gain in one month of 80,000.

The differential count of 500 cells gave: Polymorphonuclear neutrophiles, 51 per cent.; lymphocytes (the small lymphocytes being slightly in excess of the large and transitional forms), 44.6 per cent.; eosinophiles, 4.4 per cent. No nucleated cells were seen and the blood appeared practically normal.

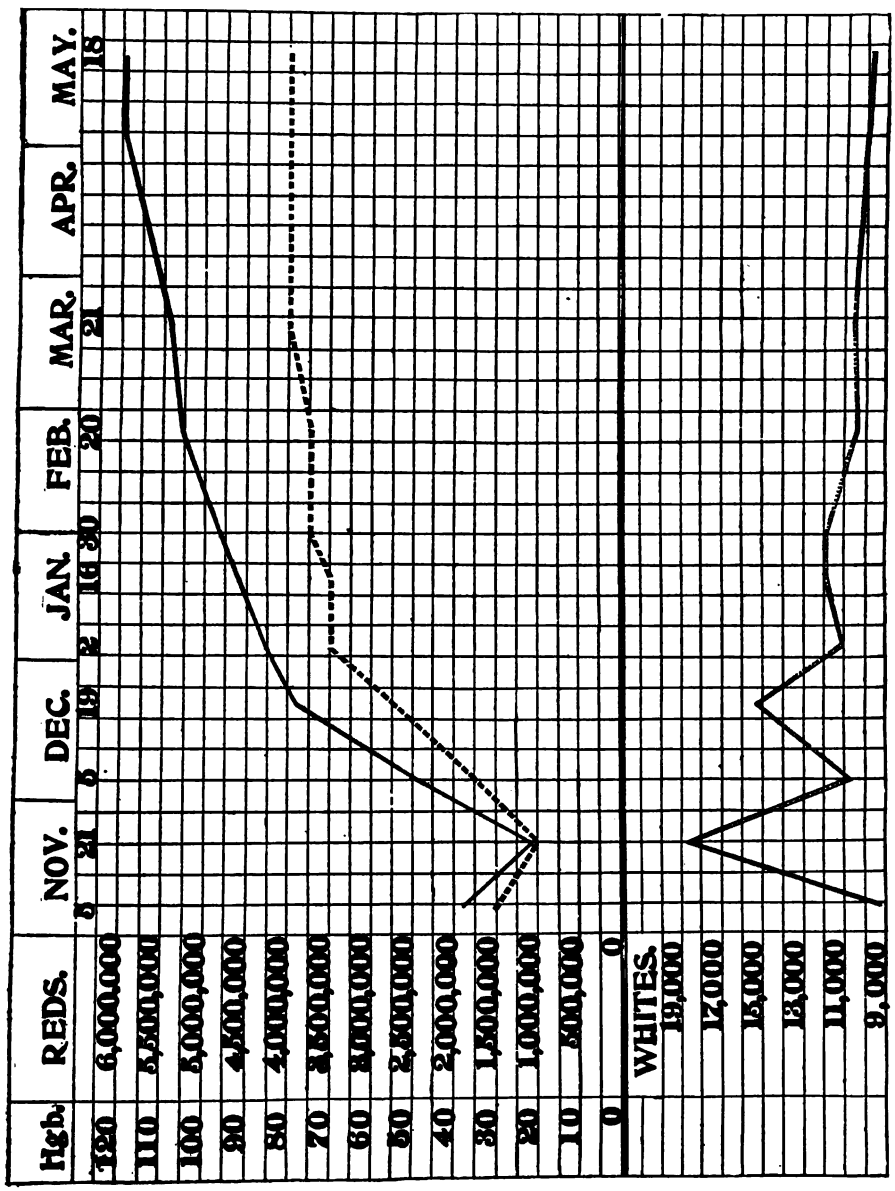
On May 18th, twenty-three weeks after the height of the disease, the examination of the blood showed: Hemoglobin, 80 per cent., a total gain of 60 per cent., but no gain in two months; white blood corpuscles, 9,700; red blood corpuscles, 5,980,000, a total gain of 4,892,000, or a gain of 508,000 in two months.

The differential count of 500 cells gave: Polymorphonuclear neutrophiles, 34.6 per cent.; lymphocytes, 64 per cent.; eosinophiles, 1.4 per cent. No nucleated red cells were seen. An occasional macrocyte and poikilocyte was seen. (See chart).

The temperature still remains slightly raised, 99°-100° F., and has only once been 98.5° F. since the onset of the disease.

In considering the diagnosis in the pernicious anemia of infancy we are forced to acknowledge that signs which are almost pathognomonic in adults lose some of their significance in infants, owing to what we may assume to be a greater instability of the blood-making function. This function, like many others in the early months of life, appears not to have reached its full development, if we may judge from the ease with which the blood reverts to its embryonic type and again recovers its equilibrium. Thus we know that megaloblasts, normoblasts, macrocytes and poikilocytes may occur in grave anemias which are not to be classed as pernicious, but we are hardly justified in reasoning from this that the occurrence of these elements are of no importance in differentiating the type of anemia. We have already pointed out the extreme rarity of pernicious anemia in infants, and the majority of cases which have been reported do not give us the data which in modern methods of blood-diagnosis are considered essential. We have failed to find reports of cases occurring in infants who showed the typical clinical symptoms and blood characteristics of an idiopathic pernicious anemia and absence of a primary cause which subsequently was proved not to be pernicious anemia. It is impossible for one safely to draw general conclusions in regard to this disease in infancy until a much larger number of cases has been studied.

The important points to be emphasized in this particular instance are: (1) The insidious onset with moderate and paroxysmal attacks of indigestion. (2) The extreme pallor and great loss of strength. (3) The absence of any possible demonstrable cause for a secondary anemia. (4) The slightly elevated temperature for months. (5) The absence of glandular or splenic enlargement. (6) The presence of the pronounced typical characteristics of the blood of pernicious anemia. (7) The absence of any considerable degree of leucocytosis. (8) The rapid improvement in the general symptoms and in the character of the blood until the infant in all respects appeared absolutely sound and healthy, which in itself is typical of the remission which often occurs in pernicious anemia. We feel that a picture so typical cannot be explained by any other diagnosis than that of pernicious anemia.



Upper black line shows number of red blood corpuscles per c.mm. Middle dotted line shows percentage of hemoglobin.
Lower dotted line shows number of white blood corpuscles per c.mm.

Great interest centers about the prognosis in this particular case. Whereas the infant is in perfect health to-day so far as can be determined, the fact remains that there has never been reported a case of permanent recovery in a well attested case of pernicious anemia in infants. Several cases of recovery in adults are on record, notably Quincke's, which also has the lowest red count ever noted (143,000 per cubic mm.), but they are exceedingly rare. We might be led by the knowledge of the greater recuperative power in the infant's blood, to hope that in a case so young as ours, the infant may resist the usual fatal tendency to a reversion to the embryonic type of blood, in the same way that it might outgrow other abnormalities of development. Speculation as to the future is, however, idle.

TREATMENT.—In regard to the treatment of these cases, little need be said. The greatest care in the management of the feeding is of first importance. Arsenic may be tried cautiously, but in our own case it was not tolerated and played no part in the gradual improvement which took place in the disease. Iron in the form of ferratin or similar preparations will certainly do no harm and may be of benefit. Cabot has recently been treating his cases with laxatives or purgatives on the theory that the primary cause lay in the absorption of toxins from the intestines, but no definite results have as yet been announced. In regard to the use of inhalation of oxygen, opinions will differ. We know of no other case in which such large quantities have been used over so long a period. The immediate and rapid improvement which followed its administration will be a strong encouragement to study its action again in similar cases in infants. At the same time, the evidence pointing to the rapid and spontaneous remission of the disease in certain cases, independent of treatment, is indisputable. It is, therefore, difficult to see how a judgment as to the efficiency of special remedies can at present be formed on absolutely scientific grounds.

DISCUSSION.

DR. WENTWORTH.—I should like to say a few words about the results of blood examination in infancy. Such men as Grawitz, Strümpell, von Noorden and others are agreed that the diagnosis of *pernicious anemia* in the adult cannot be made from a blood examination alone. The blood changes that were considered at one time to be typical of pernicious anemia have been found associated with various chronic diseases and the

deductions that can be made from the examination of blood in infancy are much less reliable than in adults. It is incorrect to say that but little work has been done on infants' blood—a great deal of very careful work has been done in the past ten years, especially in Europe. Fischl, Siegert and Loos may be particularly mentioned.

It has been found that the presence of nucleated red corpuscles in the blood in infancy has no diagnostic value but only shows the degree of anemia present. The number and size of the nucleated red corpuscles have no diagnostic significance in these cases. Differential counts of the leucocytes have no diagnostic value in the absence of leucocytosis.

It has been quite definitely settled that the cases of so-called anemia infantum pseudoleucemica are *secondary anemias* and not primary as was at one time believed.

I should like to ask whether the heart murmur persisted in this case. Hochsinger has made the statement that *functional* murmurs are very rare under three years of age. I do not know how many autopsies this statement is based upon. So far as my own experience goes I have not found cardiac murmurs in cases of anemia in infants no matter how severe the anemias were and I have made it a point for several years to examine the hearts very carefully in all the cases of anemia in infants that I have seen.

DR. KOPLIK.—The fact that the blood of infants presents the same appearances as that of adults suffering with pernicious anemia gives us no assurance as to the diagnosis. The cause of this has been stated partly by the author of the paper and partly by the last speaker, namely, that we find in some cases of secondary anemia changes that correspond very well with those of pernicious anemia in adults and still these children get well. We find nucleated red corpuscles to a large extent and of all sizes in secondary anemias with poikilocytosis and hemoglobin percentages, which are supposed to be characteristic.

A great deal of work has been done on the blood of infants and children but the difficulty is to interpret what we see and to compare the appearances with those of adults. I think for the present we do better not to make any positive diagnosis of some forms of anemia in children whether they occur idiopathically or are secondary.

DR. MORSE.—I merely wish to emphasize what the last two speakers have said, and what the reader of the paper has also said, that the rules of blood diagnosis in adults do not apply in the diagnosis of the various conditions of the blood in infancy. The attempt to apply the same rules in infancy will surely lead to errors in diagnosis. Megaloblasts and nucleated cells of all sorts are frequently found in all conditions of anemia in infancy. They are merely indicative of the tendency of the infant's blood

to revert to the fetal type and are of but little diagnostic value. The relative proportions of white cells vary from day to day under normal conditions and hence are of little value in diagnosis.

As to the classification of blood diseases I think there is no class in infants that deserves the name of anemia infantum pseudoleucemica. Cases of that disease have been described but most of them can only be regarded as examples of severe secondary anemia. A few years ago I went over the literature of pernicious anemia in infancy but could not find any cases which I was willing to accept as undoubted examples of that disease. Some may have been, but I was not convinced that they were. I hardly feel willing to say that this particular case is one of pernicious anemia, but do not dare to say that it is not. I have seen, I think, identical conditions of the blood in anemias in infants which I knew to be secondary. If we regard anemias as primary when we cannot find their cause, this case is certainly primary. It may, moreover, be pernicious. On the other hand there may be some cause for it, although it has not been found in the very careful study of Dr. Rotch and Dr. Ladd. I certainly cannot concur in the prognosis made by Dr. Cabot.

DR. NORTHRUP.—Have I misunderstood Dr. Wentworth when I thought that he said that in these cases of anemia with heart murmur there must be a heart lesion? I also understood him to say that he saw no cases of heart murmur without a lesion, and that in his experience and in that of Hochsinger, heart murmurs, without a lesion, did not occur under three years of age.

DR. WENTWORTH.—I said that Hochsinger finds heart murmurs exceedingly rare under three years of age, and I have examined the hearts of all the infants in our hospital and failed to find a murmur in connection with anemia. I have not had autopsies to verify these cases.

DR. NORTHRUP.—I should dislike to have it go on record as expressing the sentiments of this meeting that where you have a murmur the presumption is that you have a lesion, especially in patients under three years of age. I have written fully on the subject in Jacobi's Festschrift.

During the first twelve years of my medical experience I saw more dead than living children. My experience was, speaking from memory, that in the Foundling Hospital, where the children were under three years of age that we had many murmurs and no lesions. We had one typical case of malignant ulcerative endocarditis, but it was so rare that nobody recognized it before death, and by exception it serves to emphasize the rule.

DR. CHURCHILL.—I have been on the lookout for these murmurs in infants during the past few years and my observation is that they are extremely common, and if all the cases I have seen have really had cardiac disease then endocarditis is very common among children. My cases have been seen not only among hospital children but in the children of well-to-do parents. I have not followed them up at autopsy.

DR. KERLEY.—I will ask Dr. Rotch in connection with this case whether the improvement in the child's digestive condition was coincident with the improvement of the blood. In the history of the case reference is made to digestive disturbances. The child was commenced on rather a high milk formula when weaned. Could the case be a secondary anemia due to chronic indigestion and malassimilation?

That heart murmurs occur without a lesion there is not the slightest doubt in my mind. I cannot say in how many cases this has occurred. They are the cases that are diagnosed as hemic sometime during life.

DR. TOWNSEND.—I would like to ask what change was made in the food during the convalescence of the child, and I should also like to speak of two cases that illustrate what I wish to say on the subject. These two cases I have seen recently, and I think they are types of extreme secondary anemia due to the fact that the diet for a long time has been for some reason insufficient or improper for the child. One of these cases coming from a distant town had been fed on a very weak modified milk which was peptonized and pasteurized. The blood count was about to be made when unfortunately the child became much worse and died. The organs were examined by Dr. J. H. Wright, who found some slight fatty degeneration and a slight deposit of iron in the liver, not enough, in his opinion, to justify the diagnosis of pernicious anemia. I do not see how the blood count would have helped in the diagnosis in this case, owing to the embryonic character of the blood in all infants.

The other case showed the same anemia when I first saw it in a marked degree. It had been fed on laboratory mixtures, and later on home modified milk that was pasteurized and peptonized. By omitting these latter processes and gradually increasing the strength of the food the child got entirely well and has an excellent color to-day.

Now I have seen so many cases of extreme anemia in children that have been improperly fed, even where the impropriety at first sight is not apparent, that I feel sceptical about the pernicious nature of the trouble in the case reported, and should certainly not expect a relapse.

DR. WINTERS.—I would like to ask whether this baby was fed at all on pasteurized milk, whether the intestinal derange-

ments disappeared coincident with the improvement in the blood, and whether the change in the infant's food was coincident with the beginning of the improvement.

I saw a case similar to the last two reported where the baby had been fed on such milk and then improved promptly when put on the breast-milk of a good nurse.

DR. ROTCH.—It is difficult, of course, to follow a case of this kind as its history is being read. Most of the criticisms may have been answered in the paper as you will see when you come to read it. It is an unusual case, and the gentlemen who take the ground that it is one of common occurrence take an opposite position from one who is considered a high authority in this country on examinations of the blood, namely, Dr. Richard C. Cabot. The members who have spoken seemed to imply that we did not recognize the difficulty of making a diagnosis between pernicious anemia and severe secondary anemia. What has just been said about the diagnosis of pernicious anemia in childhood might apply equally well to the diagnosis of this condition in adults. You may say in either case that if you cannot find a cause for the condition, and if the examination of the blood and the clinical symptoms are typical, it is a case of pernicious anemia. Dr. Cabot carefully studied several specimens of the blood as prepared by Dr. Ladd, and gave it as his opinion that this was an unusual case which represented in every way pernicious anemia as it occurs in adults. You may say that pernicious anemia never occurs in infancy. Surely we have no right to assume such a position, and if we admit that it can exist, then the evidence is strongly in favor of this being a case of pernicious anemia.

In reply to some of the criticisms: Dr. Wentworth speaks about the heart and asks whether the murmur remains, and he implied, and I think he believes, that it was a case of organic disease of the heart. I do not believe so. In a great many cases I have found murmurs that I am sure were hemic and I think he in the future will find them also as he sees a large number of cases. In regard to this especial case I wish to say that I had unusual opportunities for studying it. I followed it from the day the child was born. At birth it had no heart murmurs and there was nothing the matter with the heart so far as I could judge by a number of careful examinations until after it developed this condition of anemia. During the anemia the murmurs developed and as the anemia disappeared the murmurs lessened and finally disappeared.

Dr. Townsend seemed to imply that he believed the child to have been improperly fed. All I can say is that according to our modern ideas of feeding I think the child was properly fed. During the first four months of life it was fed at the mother's breast on good milk as determined by examination. When it

began to have a little indigestion it was found that the mother's milk was failing, as it often does in American mothers of this type. It was then decided to wean it and it was put upon modified milk and throve. At first the milk was heated to 167° F., but later it was not. As its condition did not improve milk was obtained from another set of cows, but no change took place, and in fact the food seemed to have nothing to do with the anemic condition. Indigestion played no part in the cause of the disease, and the change to artificial feeding which I have referred to was made several months before the infant began to be so ill.

As to Dr. Cabot's prognosis he said he believed it was a case of pernicious anemia and that, while he would not say that it was impossible for babies to get well of the disease, yet in most cases they died during the first year. He fully realized, of course, the difficulty of making the diagnosis from an examination of the baby's blood. Taking into account the examination of the blood with the clinical symptoms it is seemingly a case of pernicious anemia.

SO-CALLED "CYCLICAL ALBUMINURIA," WITH PRELIMINARY REPORT OF CASE.

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Pavy[1] was the first to call attention to the condition known as "cyclical albuminuria," *i.e.*, a condition characterized by the absence of albumin in the early morning urine, by its presence in specimens passed from about nine or ten o'clock in the morning up to about eight at night, when the albumin as a rule is again absent. True, long before this time an intermittent albuminuria with a cyclical course had been recognized, but no characteristic name had been given to the phenomenon. Among the earlier writers on the subject of albuminuria may be mentioned: Vogel,[2] Ultzmann,[3] Moxon,[4] Dukes,[5] Rooke,[6] Edlefsen,[7] Furbringer,[8] Rendall[9] and Bull.[10]

Since Pavy's article and owing largely to the interest excited thereby, numerous cases have been reported, but few have been exhaustively studied. The authors of these later articles all agree in the observation that on assumption of the upright position after several hours in the recumbent position albumin appears in the urine and dwell on this fact as a causative factor. Almost all have observed also, that with prolonged rest in bed the albumin disappears from the urine, but is apt to return immediately on getting up, even after the lapse of several weeks. Many observations have been made under different conditions of life as to the effect of exertion in varying degrees in the recumbent, sitting and standing positions, of diet, of weather, etc., but beyond the association of the upright position with the albuminuria, it cannot be said that anything of importance is developed as to the true nature of the condition. Curiously enough these authors have made no exhaustive study of the urine in their cases, nor have they followed them up any considerable length of time. I have been unable to find any

post-mortem records, for the obvious reason that the prognosis is usually good.

Some interesting facts, however, have been brought out in the study of this subject. Dukes,[11] physician to Rugby School, found cyclical albuminuria in many boys and attributed it to sudden rising and the exertion of hurried dressing and running 100 yards and up a flight of stairs to attend morning prayers. These patients usually had hard, rigid pulses. with high tension and were subject to fainting spells. Dukes urges more careful urinary examinations in such cases (but not the abolition of morning prayers!)

Stirling[12] discovered the condition in 77 of 369 boys in a training-ship on the Thames, finding it more frequent and persistent among those playing wind-instruments. He attributes it to backward pressure upon the renal vessels. He suggests the term "postural albuminuria."

Rudolph[13] speaks of the prevalence of the condition in families, citing instances of his own, and also cases from Heubner,[14] Moxon,[4] Schön,[15] and Lacour.[16] He believes it to be due to an obstruction in the glomerular vessels. These, by previous inflammatory processes, have lost some of their elasticity, as a result of which they cannot prevent the leakage of albumin when subjected to the increased blood pressure of the upright position though able to withstand the ordinary pressure of the recumbent position. The frequent occurrence of some previous infectious disease and of a coincident or previous nephritis in those cases which have been most carefully studied, supports this view. The children Rudolph has seen with this form of albuminuria are sick; they are anemic, lack energy, and are subject to headache, vertigo and intestinal disturbance. He suggests that the disappearance of the albumin from the evening urine may be due to the sitting position at the evening meal and gives a table showing the absence of albumin after the mid-day meal also.

G. Johnson[17] regards a long-continued, though slight and intermittent albuminuria as pathological and serious, demanding prolonged and frequent urinary examinations. Wood[18] discusses albuminuria not due to organic disease of the kidneys, but says nothing directly about "cyclical albuminuria." He believes we may have an albuminuria due to irritation of the kidney from a concentrated urine, with the deposition of con-

siderable amounts of calcic oxalate and uric acid crystals within the renal tubules, this increased amount of crystalline matter resulting perhaps from perverted metabolism. He considers it important to detect this irritation early. He speaks of a "normal albuminuria," *e.g.*, after great muscular exertion, or a long march which, as far as he knows, is never accompanied with casts. This opinion, however, Wood expressed in 1892, six years before Darling's[19] observations on Harvard crews after the Yale race. Darling found an actual acute nephritis in many of the crew, with casts, blood and renal epithelium in the urine passed immediately after the races.

Oswald's[20] article, perhaps the most thorough one yet written, reviews the literature up to that time (1894), referring to the work of von Noorden,[21] Klemperer,[22] Rohlfing,[23] Heubner,[14] Tewes,[24] besides others already mentioned above. He gives 9 cases of his own, with reports on the amount of urine and albumin, the specific gravity and pulse-tracings (6 cases). All his subjects suffered from symptoms pointing towards renal trouble, 7 were cases of actual nephritis, the other two were doubtful. Observations on the effect of the recumbent, sitting and upright positions, with and without muscular exertion confirm the universal opinion that the upright position has the greatest effect upon the albuminuria; excessive muscular exertion and the forenoon are especially favorable to its production. Oswald frequently found nucleoalbumin in his cases and regards it as indicative of renal irritation. He attaches much importance to the character of the sediment and agrees with Leube[25] and Senator, in regarding the frequent occurrence of hyaline casts with long-continued albuminuria as a sure sign of renal disease even in the absence of co-existing organic changes. He agrees with Johnson that these cases represent a "sneaking nephritis" which may be cured or run on into a chronic form. He believes the cause of them to be a pathological condition of the glomerular tissues.

Senator discussing a case reported by Klemperer[22] speaks of a curiously interesting case; a patient with a splenic tumor always had albuminuria if he laid on his right side, never if lying on his back or left side. The albuminuria was attributed to pressure on the kidney by the tumor and carelessly observed, might have been regarded as a case of "cyclical albuminuria."

Our text-books all refer to the condition. Rotch believes in a physiological albuminuria but urges care in the examination of many specimens of urine, passed at different hours of the day, in order to exclude nephritis. Holt advises a guarded prognosis and prolonged observation of cases; if constant and long-continued, he regards it as serious. Williams believes these cases should be regarded as mild subacute or chronic nephritis and treated accordingly. Osler believes a large proportion of cases get well after a variable period but recognizes a second group when the albumin is more persistent, indicating probable organic changes in the kidney. Ashby and Wright (American edition) consider the condition not infrequent in children between the ages of eight and sixteen years. A majority improve and the albumin disappears after months or years. They consider the prognosis more serious in those with a family history of Bright's disease or a previous history of nephritis.

Various theories have been advanced to explain the phenomenon of "cyclical albuminuria." One can of course understand the presence, though not the peculiar course, of the albumin in those cases with other well-defined signs of nephritis. The most plausible explanation as to its peculiarity would seem to be those which attribute it to a combination of general and local conditions, to the combined effects of increased blood-pressure upon either altered vessel-walls or altered blood, or upon both. The increased blood pressure of the upright position must, it seems to me, be regarded as one causative factor. What is, or are, the others? Rudolph's theory seems most probable; impairment by previous inflammatory processes of the walls of the glomerular vessels, with loss of elasticity and hence greater degree of permeability with the increased blood-pressure of the upright position. von Noorden's theory, however, is worthy of consideration; he suggests a change in the blood-albumin itself in virtue of which it transudes more freely, or that we have to do with an indefinite disturbance in general metabolism[22a]. He bases the latter view upon the increased phosphates and urea found in the urine.

It must be confessed, however, that we know practically nothing of the real cause of this symptom. But whatever the cause, the actual condition confronts us; in some cases a genuine nephritis beyond a doubt—in others an albuminuria peculiar in its course and apparently without other abnormal

signs. As one peruses the literature of the subject, he cannot help asking: What becomes of them? both those without other apparent signs of organic disease, and those with evident signs. Do the women die of puerperal eclampsia? Do the men subsequently develop an interstitial process? Is this albuminuria an obscure and insidious symptom indicative of some deep underlying pathological condition, undetectable with our present methods of investigation? Or is it but a harmless condition, to be regarded lightly and dismissed with a cursory examination of urine and patient?

Weissman's theory of heredity that it is tendencies, not actual characteristics, which are transmitted from parent to offsprings is applicable pathologically, as well as physiologically. A child receives from his progenitors cells and tissues with tendencies to develop and grow in certain directions, the direction taken depending to a large extent upon the environment of the individual, using the term environment in its widest sense, to include external surroundings, general hygiene, nutrition, diet, etc. These tendencies will develop into actual conditions or characteristics obviously with varying degrees of rapidity in different individuals; thus they may begin to manifest themselves, to our imperfect eyes, in one person at fifty years, in another at thirty and perhaps in another at eleven, but in different ways and with different external signs at different periods of their development. This "cyclical albuminuria," *e.g.*, may be a manifestation of a tendency gradually developing into the actual characteristic or condition of renal cells and tissues known as nephritis, the tendency manifesting itself in one form at eleven years and in another at fifty years. To us who are working chiefly among children is this conception of the case especially of interest and importance: for in the early part of the animal existence inherited traits, both good and bad, are not traits at all—if I may be pardoned the Hibernianism—but are only tendencies capable of being moulded and markedly modified by properly-directed measures. If we can by patient study of the individual, discern early in life his vicious tendencies—for all have such—and attack them at once and persistently, we may do much to modify or even practically eliminate them, may long postpone or entirely prevent serious trouble, may prevent the evolution of a tendency into an actual condition.

I can but believe that this so-called "cyclical albuminuria"

DATE, 1901	OUNT	SP. GR.	ALBUMIN *					UREA *	CL.*	PHOS.*	SULPH*	SEDIMENT
			8 A.M.	11 A.M.	2 P.M.	4-6 P.M.	8 P.M.					
FEBRUARY—			%	%	%	%	%	%	%	%	%	
26, 2.00 P.M.	.....	1032	.....	.....	Tr.†	.....	.....	.....	.....	.....	.....	Much; many leucocytes, few blood corp. (normal), few squamous cells, 1-2 round cells (kidney), 2 f. g. casts after long search.
27, 8.00 A.M.	.....	.....	Sl. Tr.‡	.....	.....	.....	.....	.....	.....	.....	.....	As yesterday, but no casts and a few uric acid crystals.
27, 5.45 P.M.	.....	.....	.....	.....	.....	Sl. Tr.‡	.....	.....	.....	.....	.....	As this A.M.
28, 8.00 A.M.	.....	.....	0	.....	.....	.....	.....	.....	.....	.....	.....	
28, 2.00 P.M.	.....	.....	.....	.....	0.9	.....	.....	.....	.....	.....	.....	
28, 4.00 P.M.	.....	.....	.....	.....	.....	4	.....	.....	.....	.....	.....	After exercise in gymnasium, about 1 hour.
MARCH—												
1, 7.00 A.M.	.....	1019	0	.....	.....	.....	.....	.....	.....	.....	.....	
1, 2.00 P.M.	.....	1026	.....	.....	0.25	.....	.....	.....	.....	.....	.....	
2, 7.00 A.M.	380 cc.	1022	0	.....	.....	.....	.....	2.4	6.5	7	1	
2, 2.00 P.M.	185 "	1021	.....	.....	0.75	.....	.....	2.1	5.0	6.5	0.75	
2, 4.00 P.M.	50 "	1032	.....	.....	.....	1	.....	2.7	6.0	11.5	1.5	
2, 8 00 P.M.	150 "	1024	.....	.....	.....	.....	1	2.1	3.75	10.0	1.0	
Total	765 "	1024	.....	0	5	.....	.....	2.2	5.25	4.75	1.0	Total urea—16.83 grammes.
3, 8.00 A.M.	.....	1021	0	.....	.....	.....	.....	2.1	3 75	7.50	1 0	
11, 2.00 P.M.	.....	1031	.....	.....	0.75	.....	.....	2.5	9.0	11.0	1.0	
11, 4.00 P.M.	.....	1004	.....	.....	.....	0	.....	0.2	0.75	1.5	0 5	Immediately after gymnasium.
21, 2.00 P.M.	.....	1032	.....	.....	0	.....	.....	2.5	8.	8.	1.	
21, 4.00 P.M.	.....	1034	.....	.....	.....	1.5	.....	2.7	5.	13.	.....	Immediately after gymnasium.
28, 2.00 P.M.	.....	1031	.....	.....	0.25	.....	.....	2.4	8.	8.	1.	
28, 4 00 P.M.	.....	1032	.....	.....	.....	0.25	.....	2.4	3.75	11.	.....	
31, 10.00 A.M.	40 cc.	1024	.....	0.5	.....	.....	.....	2.3	6.75	5.	1.	
31, 5.45 P.M.	250 "	1025	.....	.....	.....	¼-⅛	.....	1.8	6.	8.	0.5	
APRIL—												
1, 8.00 A.M.	370 "	1022	0	.....	.....	.....	.....	2.3	4.	10.5	1.	
Total	660 "	1023	.....	.....	about	1-10	.....	2.2	4.5	7.	1.	Total urea—14.52 grammes.

14,	8.00 A.M.	200 "	1020	0	....	.....	.....	.....	2.	2.	8.	0.75	Leucocytes, ew renal cells, 1-2 uric acid crystals, 1 f. g. cast (2 slides).
14,	10.30 A.M.	100 "	1021	.....	‡Sl. Tr.	.....	.....	.....	0.9	9.	2.75	0.5	
14,	6 00 P.M.	75 "	1016	.....	.....	.....	0.5	.....	1.2	2.	3.	0.75	In house quiet until noon.
15,	8.00 A M.	190 "	1018	0	.....	.....	.....	.....	1.2	2.25	10.	1.	
15,	11.00 A.M.	95 "	1019	.....	0	.....	.....	.....	1.1	10.	3.	0.5	
15,	4.00 P.M.	85 "	1027	.....	.....	.....	1.25	.....	1.6	10.	7.25	1.	
15,	6.00 P.M.	50 "	1027	.....	.....	.....	0 5	.....	2.8	8.	6.	1.	
15,	8.00 P.M.	90 "	1019	.....	.....	.....	.....	0	2.0	3.	4.	1.	
20,	8.00 A.M.	380 "	1026	0	.....	.....	.....	.....	2.4	4.	7.	1.	
20,	9.30 A.M.	25 "	.....	.....	4.5	.....	.....	.....	1.6	7.	.....	.....	
21,	8.00 A.M.	425 "	1024	0	.....	.....	.....	.....	1.9	3.75	8.	1.	
21,	10.30 A.M.	80 "	1024	.....	1 5	.....	.....	.....	1.9	10.	5.	0.75	
23,	10.00 A.M.	265 "	1024	0	.....	.....	.....	.....	2.3	8.	11.	1.	
23,	3.00 P.M.	180 "	1016	.....	.....	0	.....	.....	1.1	6.	2.5	0.5	Bed till 5.30 P.M., then sat up one hour.
23,	5.30 P.M.	60 "	1026	.....	.....	.....	0	.....	1.7	7.	9.	1.	
23,	8.00 P.M.	55 "	1029	.....	.....	.....	.....	0.5	2.5	6.	10.	1.	
24,	8.00 A.M.	165 "	1028	0	.....	.....	.....	.....	2.4	5.	10.	1.	
24,	2.00 P.M.	180 "	1027	.....	.....	0	.....	.....	1.8	11.5	6.	.....	Bed all day.
24,	8.00 P M.	25 "	.....	.....	.....	.....	.....	0	2.9	.....	.....	.. ...	
25,	9.00 A.M.	205 "	1029	0	.....	.....	.....	.....	2.6	7.	8.	1.25	
25,	11.40 A M.	180 "	1024	.....	0	.....	.....	.....	2.0	9.	9.	1.	Bed nearly all day.
25,	3.30 P.M.	205 "	1020	.....	.....	0	.....	.....	1.7	6.	5.	0.75	
25,	6.30 P.M.	?	.....	.....	.....	... ..	0	.....	2.2	.....	.....	.....	Renal cells and leucocytes much fewer; few large round cells from bladder, 1-2 blood corp. (abnormal); 1 f. g. cylindroid tapering, with leucocytes adherent.
27,	10.30 A.M.	.... ..	.....	.....	0	.....	.....	.....	.. ...	.....	.....	.....	
29,	8.00 A.M.	205 "	1026	0	.....	.....	.....	.....	2.8	7.	11.	1.	
29,	11.00 A.M.	175 "	1010	.....	0	.....	.....	.....	0.8	4.	1.5	0.5	
29,	5.30 P.M.	325 "	1013	.....	.....	.....	0	.....	1.3	4.	4.	0.75	Bed till 5 P.M.; up in chair short time.
29,	8.00 P.M.	205 "	1007	.....	.....	.. ...	.....	0	0.4	1.5	3.	0.5	
30,	9.30 A.M.	315 "	1025	0	.....	.....	.... .	.....	2.5	7.	10.	1.	Up about 10 A.M.; walked down stairs—out to drive.
30,	3.30 P.M.	295 "	1010	.....	.....	2	.....	.....	0.6	2.	4.	0.5	5 P.M. specimen shows only slightest cloudiness with K Fe Cy solution—no reaction with heat or H N O_3.
30,	5.00 P.M.	35 "	1020	.....	.....	.....	?	.....	.....	4.	.....	.....	

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			8 A.M.	11 A.M.	2 P.M.	4-6 P.M.	8 P.M.					
MAY—			%	%	%	%	%	%	%	%	%	
3, 8.30 A.M.	.....	.....	0	.....	.....	.....	.....	.....	.....	.....	.....	Bed all day.
3, 5.00 P.M.	.....	.....	.....	.....	.....	0	.....	.....	.....	.....	.....	
8, 11.30 A.M.	120 cc.	1022	.....	0	.....	.....	.....	1.7	13.	12.	.....	Many leucocytes, singly and in clumps, renal cells more, calcic oxalate crystals. Casts: hyaline, f. g. and coarse granular, some with leucocytes and renal epithelium adherent: some of casts broader than any seen before.
8, 5.45 P.M.	165 "	.....	.....	.....	.....	7.	.....	.....	.....	.....	.....	
9, 8.00 A.M.	425 "	1018	0.1	.....	.....	.....	.....	2.1	4.	10.	.....	Leucocytes, 1-2 renal cells, 1-2 bladder cells, round; 1 coarsely gran. cast. All elements much less than yesterday's 5.45 specimen.
9, 5.30 P.M.	127 "	1019	.....	.....	.....	4.	.....	2.2	7.	15.	.....	
9, 8.00 P.M.	75 "	?	.....	.....	.....	... ..	1.5	2.1	4.	11.	.....	
10, 7.00 A.M...	345 "	1016	0	.....	.....	.....	.....	2.	4.	12.	.....	Leucocytes as yesterday, 5.45; renal cells fewer, 1-2 blood (n.), no casts.
11, 7.00 A.M.	200 "	1018	0	.....	.....	.....	.....	2.5	3.75	13.	.....	Considerable number of leucocytes, singly and in clumps. Some renal epithel. Few crystal calcic ox.; 1-2 hy. and f. g. casts.
11, 12.40 P.M.	480 "	1008	.....	0	.....	.....	.....	0.5	5.	3.	... ..	
11, 4.15 P.M.	135 "	1017	.....	.....	.....	0	.....	1.3	12.	9.	.....	
11, 6.00 P.M.	35 "	.....	.....	.....	.....	0	.....	1.6	.....	.....	.....	
11, 8.00 P.M.	145 "	1013	.....	.....	.....	.....	0	2.1	.....	.....	.....	12-40; very slight; 1-2 round cells (bladder), 1 round cell (kidney); no leucocytes, no casts.
												4.15 P.M., less than 7 A.M., more than 12.40 P.M. Few blood corp. (normal and abnormal); 1 brown-gran. cast; some granular *débris*.
12, 8.00 A.M.	265 "	1017	0	.....	.....	.....	.....	1.9	.....	.....	.....	
12, 6.00 P.M.	320 "	1017	.....	.....	.....	0	.....	1.3	9.	7.	.....	
12, 8.00 P.M.	65 "	1022	.....	.....	.....	.....	0	1.4	15.	7.	.....	
13, 8.00 A.M.	135 "	1026	0	.....	.....	.....	.....	2.5	9.	16.	.....	
13, 3 00 P.M.	260 "	1012	.....	.....	G	.....	.....	1.2	.....	.....	.....	Many crystals calcic oxalate; other elements as before, but much less in amount.
13, 6.00 P.M.	55 "	.. ...	.....	.....	.....	0	.....	2.6	6.	13.	.....	

14,	8.00 A.M...	360 "	1016	0	.....	.....	.....	.....	1.9	.....	.....	.....	Elements still less; no crystals calcic oxalate.
15,	8.00 A.M.	355 "	1020	0	.....	.....	.....	.....	2.4	8.	9.	.....	6.00 P.M. Elements, except pus, much less; pus slightly less in amount. No crystals calcic oxalate.
15,	1.00 P.M.	335 "	1009	.....	0	.....	.....	.....	0.8	2.5	4.	.....	
15,	6.00 P.M.	245 "	1016	.....	.....	.....	0	.....	1.5	6	8.	.....	
16,	5.30 P.M...	55 "	1028	.....	.....	.....	5.	.....	2.7	7.	13.	.....	Elements little increased; many crystals calcic oxalate; no casts.
24,	2.00 P.M.	418 "	1008	.....	.....	0	.....	.....	.....	.....	.....	.....	6.30 P.M. 1-2 renal cells, 1-2 leucocytes; no blood, no casts, no oxalate crystals.
24,	6.30 P.M.	280 "	1014	.....	.....	.....	0	.....	.....	.....	.....	.....	
25,	8.00 A.M...	450 "	1017	0	.....	.....	.....	.....	.....	.....	.....	.....	As last night, plus many crystals of calcic oxalate. Had a little asparagus last night.

* The percentages of albumin, chlorids, phosphates and sulphates were estimated by Purdy's method of centrifugal analysis. The urea was estimated by Doremus' ureometer.

† Tr.—Trace.

‡ Sl. Tr.—Slight Trace.

is in reality a danger-signal to be heeded and its cause carefully investigated. It has occurred to me that more extensive examination of the urine in subjects presenting this symptom may throw some light on the problem, both from the general and the local point of view, in a general or constitutional way, by showing the extent to which total excretion is going on, or to what extent and in what direction it may be abnormal; and secondly in a local way by showing, from examination of the sediment, the condition of the urinary tract. If study of a large number of cases along these lines reveal some constant departure from the normal, either in total excretion or in some particular direction, further study of that abnormality may be of service in explaining the condition. As a contribution to such a study I have the following case to report:

A. B., girl, aged eleven years, presents the following record:

FAMILY HISTORY.—Her paternal grandfather and grand-uncle (brothers) died of heart trouble at the ages of seventy and sixty-seven years respectively. Her maternal grandmother was never robust, but never had any organic disease. Her mother died in 1896 of typhoid fever, having finally an infectious nephritis; father and one brother healthy and strong.

She was originally referred to me by Dr. H. B. Favill and was first seen on January 3, 1899. Up to that time there had been nothing noteworthy in her history aside from an attack of measles in infancy. At this time (January, 1899) she had a mild attack of follicular tonsillitis, and, except for the condition of her throat, physical examination, including analysis of a single specimen of urine, was negative. Her subsequent history has been: In July, 1899, a second attack of measles; in August, 1900, while on Cape Cod, an irregular fever, with intestinal symptoms confining her to bed for twelve days, said by attending man to have been typhoid; in October, 1900, on her return to Chicago, an attack of intestinal catarrh; vaccination in April, 1900, and again on January 27, 1901, both times with negative results. Several examinations of the urine made at intervals during the last two years have shown nothing abnormal. I did not attend her during her intestinal attack in October, 1900. On February 26, 1901, I saw her again, with a slight attack of follicular tonsillitis. Physical examination then showed a remarkably well-developed and well nourished girl, with good color, and except for the characteristic signs in the throat nothing of special

interest until the urine was examined. The results of this examination are recorded in the table. I attached but little importance to the analysis of this specimen, as I have often seen albumin with other signs of renal irritation in tonsillitis as well as in other infectious diseases. As the urine did not immediately clear up, however, I began to make systematic observations and soon discovered that the albumin was running a so-called "cyclical" course and that, furthermore, the sediment showed the signs of serious organic disease. We will return to a more detailed study of these observations later.

Repeated physical examinations made during the last three months have shown the following: *Development, nutrition* and *color* remarkably good; *mental condition*, bright and happy; *chest*, slight increase in the area of cardiac dulness to the left, apex fifth space in mamillary line; heart action regular, normal vigor, no murmurs; lungs negative, except as mentioned below; *abdomen* negative; limbs and extremities negative. No vaginitis, no leucorrhea, no intestinal parasites either in stools or about vulvæ. *Pulse*, normal force and vigor, never irregular, never intermittent, rather rapid, 84 lying down, 96 sitting, 112 standing.

March 2d—Blood, red, 4,860,000; white, 11,000; hemoglobin, 80 per cent.; 2 P.M., immediately after lunch.

April 15th—Height, 4 feet 7 inches; weight, 86 pounds.

The urine has shown quite constantly the presence of albumin between the hours of 10 A.M. and 8 P.M., and its absence in all morning specimens with but two exceptions. The sediment has shown the presence in varying amounts of renal and bladder epithelium, pus, blood, casts and crystals of calcic oxalate.

There should be mentioned, in addition to the above record, the occurrence of a dry pleurisy (left) from April 20th to 25th, and a small abscess of the gum early in May (3d to 9th). The absence of all symptoms and signs of discomfort at all other times has been a striking feature of the case. The child has been to outward appearances perfectly well. She has been up and about, except at the times noted in the table; has been to school, to gymnasium, and up to about the last of April has led the life of a healthy child of her age.

The above record reveals a family history of cardiovascular trouble, a previous history of several attacks by infectious diseases, some possibly of a rheumatic nature, and finally a urine

showing the existence of actual organic disease with a cyclical course to its albuminuria, this organic disease being absolutely without any subjective symptoms directly referable to it.

The chief interest of the case of course centers in the condition of the kidney as manifested by the urinary examinations, and to a study of these examinations I would now invite your attention.

Eighty specimens of urine have been examined, passed at different hours of the day between 7 A.M. and 8 P.M., the observations extending from February 26, 1901, to May 24, 1901. Looked at as a whole the child is found to be passing a fair amount of urine, the average of eight complete days being 722 c.c. It is as a rule rather concentrated in character and shows a high specific gravity and percentage of urea. Total excretion as a whole is not impaired and the kidneys are performing well their function of elimination, a fact accounting for the entire absence of even the mildest symptoms of uremia, such as headache, general lassitude, etc.

We note the character of the sediment, which together with the albumin leaves no doubt of the organic nature of the disease. The reaction has been almost invariably acid, indican has never been found in excess, sugar has never been found at all.

Curious indeed and most interesting to watch, have been the fluctuations in albumin. The excessively high percentages have invariably been traceable to some definite specific cause. The 4 P. M. specimen of February 28th for example, was undoubtedly due to a too early return to the gymnasium after her attack of tonsillitis. Again the 9.30 A. M. specimen of April 20th, with 4.5 per cent. of albumin was coincident with her attack of pleurisy. She was put to bed at this time and note the diminution and disappearance of the albumin during the following two days, to return in a smaller amount (5 per cent.) in the evening specimen of April 23d. On this day she sat up from 5 to 6 P. M. to have her bed made. After that she stayed in bed till the 30th, arose at 10 A. M., walked downstairs and went out to drive. The 3.30 P. M. urine showed 2 per cent. of albumin. On May 8th, after several days in bed with a slight abscess of the gum and cervical adenitis, she arose at noon and at 5.45 P. M. voided a specimen with 7 per cent. of albumin, so great an amount indeed that it continued to "leak" through

the night, the next morning (May 9th), urine showing .1 per cent. Nor did the system and kidneys regain their equilibrium throughout the day, for we find the 5.30 P. M. specimen with 4 per cent. of albumin and the evening specimen with 1.5 per cent. Although the child's general condition at this time seemed excellent, I decided to put her to bed for at least two weeks, such was the character of the urine. Note the result in the record from May 11th to 15th inclusive. On May 15th in consultation with Dr. Favill, it was decided to segregate the urine on the 17th. That the kidneys might be acting under every day conditions, I had the patient get up on the afternoon of the 16th. Note the 5.30 P. M. specimen, 5 per cent. of albumin. The 8 A. M. and 11 A. M. specimens of May 17th contained no albumin. The segregation, however, was indefinitely postponed, such was the objection of the family, hence I am unable to report whether the child has a unilateral or bilateral process going on. The patient has now been in bed practically for two weeks and the analyses of May 24th and 25th are as per table.

The effect of exercise upon albuminuria has been by no means constant. I have already referred to the 4 P. M. specimen of February 28th, with its 4 per cent. of albumin after an hour in the gymnasium. That this high percentage was not due to exercise alone is shown by the record of March 11th under corresponding conditions: no albumin at all. I cannot account for the quality of this specimen with its low amount of total solids. The urine of March 21st after gymnasium work showed 1.5 per cent. of albumin.

Further detailed study of the table shows departures from normal excretion in certain directions aside from the albumin and sediment. The high percentage of urea has been mentioned but cannot be regarded as excessive considering the age of the child. The chlorids as a whole range low, 49 out of 60 specimens examined, or 83 per cent., being below 10 per cent., while the phosphates range high, 36 specimens or 62 per cent. being 7 per cent. or over, 22 under 7 per cent. The sulphates are about normal.

Does the albuminous urine differ markedly from the non-albuminous in other characteristics? In the matter of specific gravity, it cannot be said there is any marked difference, it is, as a rule, high in both groups. That a large amount of albumin does not necessarily mean a high specific gravity is evi-

denced by the 3.30 P. M. specimen of April 30th, which with 2 per cent. of albumin has a specific gravity of 1010, this low specific gravity being due to the low percentage of urea, only .6 per cent.

Do the percentages of urea, phosphates and chlorids range differently in the two groups? Regarding 2 per cent. as the dividing line between high and low urea, we find that 13 of 22 albuminous specimens, or 60 per cent. are high, and 9, or 40 per cent. low, 21 of non-albuminous specimens, or 52 per cent. are high and 19, or 48 per cent., low. In other words the proportion of high to low urea is slightly greater in the albuminous than in the non-albuminous urines. The chlorids, as has been noted, run low and the proportion of low to high is about the same in both groups. Eighteen of 20 albuminous specimens, or 90 per cent., being low (less than 10 per cent.); 24 of non-albuminous specimens, or 85 per cent. being low; the phosphates resemble the urea in running high; 13 of 20 albuminous specimens, or 65 per cent., are high; 23 of non-albuminous specimens, or 70 per cent., are high. The sulphates show but little variation, ranging as a rule from .5 to 1 per cent., and this range I have found constant in many other cases both in health and sickness.

The relation of sediment to albumin is always constant; the greater the amount of albumin, the more the sediment; casts, pus, blood, renal epithelium and crystals of calcic oxalates were found in non-albuminous specimens, but usually in smaller amounts. Whatever the immediate or exciting cause of the albuminuria it evidently acted to produce an increased exudation of leucocytes and desquamation of epithelium in the kidney. Increase in blood-pressure might explain these phenomena.

On the whole, the albuminous urine cannot be said to differ markedly from the non-albuminous except in the amount of sediment. Both groups are somewhat alike in departing from the normal in the matter of urea, phosphates and chlorids, the first two being above the average, the last below it.

It will thus be seen that this case with its condition of "cyclical albuminuria" is passing a urine which shows excretion, as a whole, well-maintained, especially in the matter of urea, but which departs from the normal in the amount of phosphates and chlorids eliminated. Beyond this simple statement of facts but little can be said. This is only a single

case and we cannot say whether the increased phosphates and diminished chlorids are a peculiarity of this one subject, or are characteristic of cases with "cyclical albuminuria" generally.

As regards urea and phosphates, however, the observations here are in accord with those of v. Noorden. Future investigations will doubtless settle these relations, but until such time it is not worth while to discuss the questions naturally occurring to us: Are the conditions causing an abnormal excretion of phosphates and of chlorids in the urine well-known? Do these conditions tend to produce a nephritis, either immediately or remotely? If so, can we prevent their development or modify them once established? Will they explain the phenomenon of "cyclical albuminuria"?

The origin of the nephritis in this case cannot be determined with any degree of certainty. It doubtless has arisen from one of the several infectious attacks which the child has had, viz: measles, tonsillitis and intestinal. In this connection the following reports are of interest, the one from a bacteriological examination of the urine (2 P.M., May 23d, a non-albuminous specimen) the other from a drop of blood subjected to the Widal test:

Urine.—"Cover slips from the centrifugated sediment show short bacilli, cocci in chains, and a very small diplococcus. No tubercle bacilli could be found in a large number of specimens. Plate cultures gave the same forms of organisms as seen in the sediment. None of the colonies of bacilli gave the Widal reaction with typhoid serum, and a colony cultivated gave all the features of the B. coli communis. The streptococcus is not the streptococcus pyogenes. The bulk of the sediment was inoculated into two guinea-pigs, one intraperitoneally, the other subcutaneously, the former dying promptly from peritonitis. The other will probably live."

As the specimen was not obtained by catheter we cannot attach any significance to the presence of the B. coli communis. The completeness of the bacteriological report, however, is of value in excluding a tuberculous process in the kidney.

Blood.—The report from this investigation reads "incomplete reaction," with the oral statement "as if the patient had an abortive form of typhoid or had had the disease within the last few years."

These reports thus prove nothing as to the origin of the nephritis, though that from the blood tends to confirm the diag-

nosis of typhoid last summer. But the very obscurity of cause in this case simply emphasizes the importance of urinary examination in all infectious diseases.

Until recently but little treatment has been adopted, except to watch the urine closely and to insure a generally hygienic life. She is now on a diet low in nitrogenous material, is urged to drink much water, and at the suggestion of Dr. Green, of St. Paul, who saw the case in consultation on May 17th, is receiving urotropin gr. v., t. i. d. She is having massage and light Swedish gymnastics.

The marked improvement in the character of the urine leads one to hope that eventually complete repair will take place in the kidney. The case, however, must be carefully watched, especially at critical periods, *e.g.*, these next few years with the disturbing influences of puberty, during pregnancy and labor, or on the occurrence of any infectious disease. At such time the urine should be examined, and needless to say, more than one specimen and under different conditions of life; we shall not then be "caught napping" or be surprised by an uremic convulsion "like lightning out of a clear sky."

I have no conclusions to offer from this study of a single case. Perhaps, however, the child's record serves to emphasize the importance of urinary examination in all our cases, and furthermore of several specimens of urine passed at different hours of the day under varying conditions of life. The necessity of examining the sediment of non-albuminous urine is also evident.

I am indebted to Prof. W. S. Haines for examination of several specimens of urine; to Dr. S. G. Wells for the bacteriological report on the urine and to the Bacteriological Laboratory of the Chicago Board of Health for the report on the blood.

460 DEARBORN AVENUE.

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DISCUSSION.

DR. DORNING.—This is a very interesting subject that has been brought before us and should not be dismissed without some consideration. I have in mind one case in which twenty years ago albumin appeared in the urine once a week, invariably on Sunday. In the course of a year it appeared every third or fourth day, and was then often associated with excessive exercise. Some five years later this patient developed a cough with expectoration, but at no time were tubercle bacilli found in the sputum. Eight years later bladder symptoms appeared. On cystoscopic examination a small linear ulcer was found at the base of the bladder. For some time after the cystoscopic examination the urine was loaded with pus. At no time were casts found. Repeated examinations for tubercular bacilli gave negative results. Three or four guinea-pigs were inoculated with the urine: one died in two weeks, and the others were killed later, but none of them showed any evidence of tuberculosis. At present the patient does not cough and only the physical evidence of a small adhesion at the left apex can be detected. The urine still contains albumin most of the time but no casts.

Another remark that I would like to make concerns the movability of the kidney in this particular class of cases. Occasionally a movable kidney seems to account for a transient albuminuria, and sometimes albuminuria and casts. All of these symptoms have disappeared in some reported cases of movable kidney after a fastening of the organ by operation. It has been noticed that palpation of a kidney has been followed by albumin and casts in the urine subsequently voided, whereas it was not present previously. I think it would be well worth observing, in children especially, whether vigorous palpation of the abdomen as practiced in some of our examinations, does not give rise to a transient albuminuria: in such an event it would be the result of a traumatism.

DR. CHURCHILL.—I would like to ask in regard to that case under observation for twenty years at what time of the day the albumin appeared.

DR. DORNING.—On Sundays it appeared invariably in the afternoon.

DR. PACKARD.—Dr. Dorning's remark about movable kidney reminds me of a case that was sent to me on account of a large amount of albumin in the urine. I found that there was always a large amount in the evening, but little or none in the morning, and physical examination showed a movable kidney. When I get specimens of this patient's urine for examination I can always tell when the patient's pad is out of place by the presence of albumin. Whenever we have a large amount of albumin in the evening urine and a small amount or none at all in the morning we should look with especial care into the question of the presence of a movable kidney or of stone in the kidney.

I would like to ask whether in Dr. Churchill's case, the albumin found was entirely serum albumin.

DR. WINTERS.—I had in the case of a girl twelve years of age, in spite of every care as to exercise, etc., albumin appear regularly though she never gave any other evidence of kidney disease. At one time she was kept in bed for four months, on a milk diet for two months of that time, and no albumin appeared during the entire period. Very soon after getting out of bed albumin appeared again and I then advised her father to send her to California and she has now been there two years, living during the entire period as other school girls do, and there has been no reappearance of albumin.

DR. KOPLIK.—I have under observation such a case, the daughter of a physician. I gained the impression from this case and from the literature that all these patients have to be

watched very closely. In these cases there may be a very mild nephritis, and this was the condition probably in my case. This girl began with an albuminuria, cyclical in character appearing after exercise at intervals of a few months, with occasional casts. She was dieted very carefully and the albumin and casts have disappeared, but it is difficult to say when they may reappear. In this case the albumin was so slight at times as to be scarcely seen except with the most delicate tests.

DR. CHURCHILL.—As to the question of movable kidney, I examined this child carefully with that point in view but did not find one. The albumin was serum albumin.

PROBABLE ETIOLOGY OF RECTAL POLYPI IN CHILDREN.

BY FRANCIS HUBER, M.D.,
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In the different works on diseases of children, the varieties of rectal polypi are described, their situation is pointed out, and the appropriate treatment is given. Little, however, is said regarding the etiology, nor is any attempt made to elucidate the question. A similar result is noted, when the special works upon rectal disorders are consulted.

We are told the etiology is unknown. Many of the authors entertain the view that the affection or a predisposition to such is congenital. Some assert that mechanical causes may accelerate the growths or by causing superficial lesions give rise to secondary inflammatory processes. One author, Hauser, because of their uncertain origin, is inclined to attribute (at least the multiple form) to the action of certain microorganisms. The polypi are generally met with at points exposed to frequent irritation. The location or "loci minoris resistentiæ" being the cecum and rectum—for the feces are apt to accumulate in hardened masses in the lower bowel and an increased pressure is found to take place immediately above the ileocecal valve.

The obscure nature of the subject led me to send a note of inquiry to several prominent specialists, for definite details and individual views. Among the replies received, the following from Dr. J. P. Tuttle, of New York, represent the current views:

"The question of etiology in the production of rectal polypi in children is a very difficult one. One must distinguish in the beginning between the different varieties of polypi. Writers upon this subject are in the habit of calling everything a polypus that has a pedicle. As a matter of fact we may have benign and malignant polypi. The large majority of those occurring in children are of the adenoid variety. These may begin as flat sessile tumors, and by their weight and the expulsive action of

the intestine be dragged downwards until a pedicle of mucous membrane is formed.

Heredity undoubtedly has some influence, as was claimed by Esmarch many years ago. Inflammation also may cause the development of these tumors.

The action of certain parasites, such as the distoma hematoba, and microbic infection of the adenoid tissues may also have an etiological influence.

In the other varieties of polypi, such as the mucous polypus, which occurs occasionally in children, one may say that it is a malformation. In the fibroid polypus it is a question whether this is a result of the intrauterine inflammatory process or a malposition of the fibrous tissues. Inasmuch, however, as most all of these tumors have been found at birth or shortly thereafter, one may hardly attribute any etiological influence to intestinal infection by ingested materials."

In the discussion let it be understood that we are only concerned with the benign type met with in children, the growths presenting themselves in one of two forms. (1) The soft gelatinous, composed of the elements of the mucous membrane, and (2) the mixed variety supplemented by the cellular tissue beneath, the hard or fibrous polypi.

SYNONYMS AND DEFINITION.—Adenocele, adenoma, or adenoid tumor is defined as an hypertrophy of gland texture. The term polypus is used synonymously with the above, since all these growths upon the secreting surface for the most part contain glandular elements. In the further study of the nature of the growths, we must consider the anatomical elements of the rectal mucosa which contains villi, solitary follicles and tubular glands of Lieberkuhn. These structures must be studied in order to explain the origin and development of the polypi.

Polypi, common in the lower rectum, though met higher up, usually originate within an inch or an inch and a half from the anus and are attached by a pedicle varying in length and thickness. If of the soft variety, they may be villous or glandular. The villous vary in size from a pea to a Concord grape and are pedunculated; the glandular or cystic form has a shorter and thicker pedicle. This form is apt to undergo malignant transformation. Both bleed freely. In the mixed variety, the polypi are hard and the tumor contains mucous elements and submucous cellular tissue. The importance of bearing in mind

the fact that the growths contain the constituent elements of the mucosa and submucosa will be appreciated later on.

Usually single, they are not infrequently multiple in character. Mr. Thomas Smith has recorded three cases of disseminated polypi of the adenoid class in young persons. Cripps also has reported cases of multiple polypi springing from the rectum and colon. Kammerer reported (ARCHIVES OF PEDIATRICS, April, 1899) a very interesting case, a girl about six years old with multiple growths in the rectum and colon. Those in the latter situation were removed after the abdomen was opened. He also refers, in a brief way, to a previous case occurring in a girl eighteen years old, with hundreds of smaller growths. Dr. A. Vadjo (*Jahrbuch für Kinderheilkunde*, Bd. L., p. 411) discusses the subject exhaustively in an excellent article.

A number of cases have come under my observation, in which two or three were found in the lower rectum. In a few instances occurring in members of the same family; in two others, the patients were cousins. Before we enter upon any detailed discussion, a consideration of the following case is suggested.

Michael D., six years old, of German-American parentage, undersized and somewhat backward, for years has suffered with recurring attacks of bronchitis and asthma. Has had pneumonia a number of times.

Physical examination.—Mouth breather due to adenoids; chest somewhat "barrel-shaped"; moderate sized goitre. Treatment: nasal irrigations were ordered and for his asthmatic attacks five grains of iodid of potash three times a day with tincture of belladonna was followed by some improvement. As the treatment gave relief for the time being only, it was decided to give him free nasal breathing, hoping that the tendency to new catarrhal processes would be lessened. After the adenoids were removed his asthmatic attacks were reported as being milder and less frequent. A further interesting observation was made. It was noticed that when he lived in New York there was a complete cessation of the paroxysms, and during the months that he remained in this city he had complete relief. Upon returning to Brooklyn the attacks would recur, but were milder in character.

A further point of interest, and one which interested us a

good deal, was the frequent complaint of pain and itching in the anus, with tenesmus and some blood. There was no diarrhea nor other evidence of proctitis or dysentery. A rectal examination revealed a few small flat polypi and several eminences in the mucous membrane. Cold water injections were ordered and when regularly carried out gave him a great deal of relief.

To recapitulate—an undersized boy, somewhat backward, with pronounced aprosexia due to adenoids. In addition goitre and moderately enlarged lymph nodes furnished further evidences of the status lymphaticus. The abnormal rectal condition was doubtlessly a local manifestation of the same nature. It may be stated that these small growths are not infrequently the source of a persistent anal pruritus in such patients.

The case is reported to serve as a text for some very interesting observations and conclusions as to the probable etiology of rectal polypi. In this instance and in quite a number that have come under observation in the past four or five years, I have noticed one feature common to all the cases—*rectal polypi were only found in patients who at the same time* showed evidences of lymphoid hypertrophies in the nasopharynx with other manifestations of the constitutio lymphaticus, status lymphaticus.

This can hardly be a mere coincidence. On the contrary, the observations made in my own cases and in those in the practice of professional friends, have led me to believe that the variety of rectal polypi under discussion is simply a local manifestation of the status lymphaticus.

Through the courtesy of Dr. H. Koplik * I was allowed to examine the interesting case reported by Kammerer. Both tonsils were enlarged and adenoids were found. Recently Dr. Jos. Huber saw two cases within a week. He writes: "Have looked up the two patients (cases of rectal polypi); first, age nineteen, female, turbinated hypertrophies and mild adenoids; second, age seven, male, adenoids very marked."

About the time of writing Dr. MacHale directed my attention to a case at the Vanderbilt Clinic in which, upon making a digital examination, a polypus was torn away and escaped with some blood when my finger was removed from the rectum. In

* Dr. Koplik writes me that he had treated the child on several occasions for attacks of tonsillitis.

this case two large adenoids were present in the nasopharynx. Two cases in girls about sixteen or seventeen who had been under treatment years ago are recalled, both having suffered from rectal polypi, and in both cases the tonsils were enlarged.*

So much for clinical observation. It may now be interesting to study the question from another standpoint—that of the pathologist.

In Lilienthal's case reported as hyperplastic colitis and to cure which the entire colon was removed, the pathological condition was found to consist in a hyperplasia of the solitary lymph follicles (*Medical Record*, February 2, 1901).

Wm. M. Beach concludes that the growths "contain the constituent elements of the mucosa and the submucosa" (*Philadelphia Medical Journal*, December 16, 1899).

S. G. Gant, in an informal discussion, speaks of the similarity of the microscopic appearances of nasopharyngeal adenoids and rectal polypi.

Vadjo reports a case with autopsy and described the lesion as follows: "Das wesen der Krankheit wird durch circumscripte und multiple, eine grössere Partie des Darmzuges betreffende, primäre Drusenwucherung bedingt."

In the dyscrasia to which the term "status lymphaticus" has been applied, we have in addition to the characteristic changes in the nasopharynx, among other lesions, the following bearing directly upon the subject; the abdominal lymph nodes, especially those of the intestines and mesentery are strikingly enlarged. The Peyer's patches and their follicles and the solitary follicles project prominently above the surface of the mucosa. The swollen mesentery nodes may remain discrete or form a solid mass of lymphatic tissue. The nodes of the entire gastroenteric tract are frequently involved in the hyperplasia.

Dr. Bovaird, in speaking of a specimen presented at the Section on Diseases of Children of the New York Academy of Medicine, said: "The only change of note, as far as the colon and rectum were concerned, was the hypertrophy of the solitary follicles. These were enlarged throughout the whole extent of the large intestine, the enlargement being somewhat more marked in the upper part of the colon. The enlargement was fairly uniform and of such extent that the individual follicles

*Since the above was written, Dr. C. kindly referred to me a girl about fourteen who had been troubled with rectal hemorrhages for some time. She was undersized and a confirmed mouth breather. A digital examination revealed a solitary rectal polypus upon the posterior wall.

stood out as distinct, small grains, upon the background of the otherwise normal mucous membrane. The walls of the gut seemed unchanged."

To sum up briefly, then, it is fair to regard the rectal polypi under discussion as to the local manifestation of the status lymphaticus. In all the cases of rectal polypi which have come under personal observation or which have been observed in the practice of friends, Waldeyer's tonsillar ring was involved. In the two conditions there is a hyperplasia of tissue—in one, of the lymphoid structures of the nasopharynx, in the other of the intestinal solitary follicles. Both these hypertrophic states are but part and parcel of the general lymphoid hyperplasia, developed to a greater or less degree in the "conditio lymphatica." Secondly, the pathology, as far as the large intestine is concerned, is identical in status lymphaticus and in the case of the polypi.

Whenever the follicles are enlarged constipation, diarrhea, or some peculiar idiosyncrasy in the patient, induces a further increase in their size. Intestinal peristalsis and the passage of fecal masses exert a downward traction, gradually forming a pedicle and not infrequently a tearing away of single growths.

The question now arises, if the conclusion be correct—Why are polypi not more common?

The large number of cases of adenoids apparently without any polypi in the gut requires an explanation. Many of the patients present rectal symptoms, hemorrhages slight in amount, pruritus, fissures, mucorrhea with or without tenesmus and even prolapse. A digital examination reveals either small—more or less distinctly pedunculated growths or simple distinct elevations corresponding to the enlarged solitary follicles. The frequency with which even large polypi are overlooked is well known. All writers refer to the fact that polypi are not infrequently torn from the walls by the fecal mass and passed with the contents of the intestines.

Reference has already been made to the opinion held by some, that the affection or a predisposition to such is congenital. In this connection we may refer to the suggestive and pertinent views of Dana. He has directed attention to the teratological defect or weakness or occasional unequal vitality of the different tissues of the body, particularly observed in the study of nervous diseases. The importance of the subject leads me to quote his conclusions.

"In the study of nervous diseases one becomes impressed

with a fact which does not seem to have been appreciated by pathologists in their studies of pernicious anemia. This is the occasional unequal vitality of the different tissues of the body. For example, in the muscular dystrophies we know that the muscular system is congenitally short-lived and wastes away before the individual's other organs are even matured. In hereditary chorea the gray matter of the brain cortex, especially in the central convolutions, dies before the man is forty. In certain forms of primary dementia the same fact is true. In locomotor ataxia and the spinal atrophies and other degenerative diseases, certain systems are born with a weak vitality and succumb to infections, toxins, or accidental injuries, which do not affect the other parts. Of the millions who get syphilis only some thousands have tabes, and of the thousands who are poisoned with lead only a few have an atrophy of the cornual cells. The same law, I believe, applies to other structures. The kidneys and the liver atrophy and die early in some, while others subjected to the same influence continue healthy. These tissues have inherited a feeble resistance to deleterious agents."

The factor of teratology or unequal vital endowment of the tissues is one of the most important and compelling in the explanation of progressive degenerative diseases of the human body, and it appears applies equally to the subject under discussion. In the dyscrasia underlying the changes in the nasopharynx and large intestine the addition of a local cause in the latter, as diarrhea or constipation or an individual idiosyncrasy would tend to increase further the size of the solitary follicles. The weight, together with the expulsive action of the intestine would tend to drag them downward until a pedicle of mucous membrane is formed, and thus the polypi are completed.

A CASE OF CHRONIC ARSENICAL POISONING IN AN INFANT OF SEVEN MONTHS.

BY JOHN LOVETT MORSE, A.M., M.D.,

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Chas. W. was born August 15, 1900, about three weeks premature. He was the first child and weighed five pounds. He was normal at birth but an hour or two later became blue and collapsed. As it was warm weather he was not placed in an incubator but was put at once into his basinette. This was lined with blue sateen and had a canopy of the same material. He was fed from the beginning on modified milk from the Walker-Gordon Laboratory.

He did not seem vigorous from the first. He was always more or less blue about the mouth and eyes, and at times became very blue. Although the heart was examined repeatedly nothing abnormal was detected until September 30th, when a faint systolic murmur was heard at the apex. Although his digestion was feeble yet it steadily improved from the beginning and after the first few weeks he gained steadily but slowly. His general condition improved rapidly, although the blueness about the mouth and eyes was always present. The systolic murmur was heard at intervals.

About the first of February he ceased to gain and lost his appetite. About the middle of February he began to lose color rapidly and by the first of March the pallor was very marked. The digestion at that time was normal but there was no gain in weight. The cyanosis was rather more marked and the systolic murmur was louder. There was a slight rosary. The anterior fontanelle was decidedly large. Physical examination was otherwise negative. It was then thought that he might be suffering from the lack of fresh food and he was therefore given a home-modified milk, unpasteurized.

Two or three days later he began to pass his urine often but in small amounts. It stained the diapers red. On examin-

ation the urine was reddish, acid, 1012 and contained a trace of albumin. It contained a little normal blood, an occasional blood cast and hyaline cast with blood adherent. The eyelids were a little puffy. Physical examination was otherwise as before.

A venous hum soon developed in the neck. The urine continued reddish and contained a large trace of albumin and considerable blood. At this time, March 8th, Dr. Ogden found a trace of arsenic in the urine.

Almost everything in the room was at once examined for arsenic. The blue sateen with which the basinette was lined contained a trace of arsenic. Nothing else contained an appreciable amount. There seemed no doubt that the source of the arsenic in the urine was to be found in the blue lining of the basinette. He was taken out of the basinette March 12th and put into another room, in which there was known to be nothing containing arsenic.

In spite of the change of food he had not up to this time regained his appetite and had not gained in weight. He was even paler and the systolic murmur in the heart and the venous hum were louder. The spleen, which was not palpable the first of March, was now easily felt at least 2 cm. below the costal border. There was no puffiness of the eyelids and no nasal discharge. The gastroenteric tract was normal. The urine was increased in amount, probably because of the ingestion of much water, but continued reddish and contained a large trace of albumin, considerable normal blood and a few casts.

He began to improve within a few days after he was moved into the other room. His appetite slowly returned and his color began to improve. He began to gain in weight during the last of March. The murmur in the heart was gone April first and the venous hum about the middle of April. The spleen was not palpable after the first week in April. The urine gradually cleared up until about April 20th; it was then pale and not reddish, contained only a slight trace of albumin, a very little blood microscopically but no casts. The gain in weight had been progressive but not very rapid up to this time. The gain in strength and color had been more marked.

At this time the baby had a severe coryza as part of a house epidemic, the temperature rising to 104° F. The urine immediately became red, the albumin increasing to at least one-eighth per cent. The sediment contained much normal blood and a

considerable number of hyaline, fine granular, epithelial and blood casts. Recovery from the cold was rapid. The trouble with the kidneys has continued, however, although no symptoms referable to it have developed. The recurrence of the renal complication has unfortunately prevented the re-examination of the urine for arsenic.

NOTE.—August 17, 1901—The urine became normal about the middle of June. Improvement has been rapid and uninterrupted since that time.

ACUTE RECURRING RESPIRATORY FAILURE IN THE NEWLY BORN, WITH SYMPTOMS APPARENTLY OF BULBAR ORIGIN.

BY IRVING M. SNOW, M.D.,

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I have had two extraordinary experiences with newly born infants which are certainly worthy of record. Both children were born after a normal labor, and for the first few days of life were in perfect health. Suddenly they developed a peculiar group of symptoms, the chief of which was a failure of respiration associated with opisthotonos.

In the *Lancet* of November 24, 1900, Dr. George Keith describes a fourteen and one-half hour struggle to save the life of a boy eight days old. The baby suddenly stopped breathing and for hours was kept alive by inhalations of oxygen and artificial respiration.

The history of Dr. Keith's case coincides so exactly with the symptoms of my own two patients that I should consider all three patients as affected with the same malady which caused the death of one child and a nearly fatal issue in the other two.

Inasmuch as a description of the condition and its treatment is to be found in no treatise on diseases of children, and as some physician might give up in despair a case which persistent work might save, it may be of interest to relate the clinical history of the three infants.

CASE I.—D. W., born August 27th after a normal labor—first presentation; forceps used for fifteen minutes to terminate second stage. There was no obstetrical injury, no asphyxia; weight at birth seven and one-half pounds. The baby throve with all functions in order until the fourth day of life, when he suddenly grew cold, blue, and the breathing became shallow and irregular. When I saw the case with the accoucheur, the child was badly cyanosed, with gasping respiration. There

were harsh râles through both lungs, heart sounds normal, rectal temperature 100.6°. For the next nineteen hours, until seven in the morning, the baby showed the most alarming symptoms, which caused us to expect death at any moment. The symptoms were: (1) The frequent stoppage of respiration with intense cyanosis and no spontaneous effort at natural breathing; (2) the frequent occurrence of convulsions which seemed closely associated with the respiratory pause; (3) intense exaggeration of the pharyngeal and anal reflexes. Thus when the intestine was irrigated or food or medicine given by rectum, the child grew rigid and ceased breathing. The same apnea occurred when liquid food and medicine were placed in the mouth. This, of course, precluded the giving of nourishment and all usual means of treatment.

The most striking feature of the illness was the apparent inability of the baby to breathe for itself. Life seemed to be sustained only by artificial respiration and the continuous administration of oxygen. If the artificial respiration was suspended for from five to fifteen minutes, the breathing would grow slow and superficial and would finally stop; the heart's action became weak and irregular only when the cyanosis became extreme. Throughout the afternoon and night the baby had frequent attacks of opisthotonos. The eclampsia was often independent of the respiratory disturbance. Occasionally we could establish respiration for a while, but it would always fail and finally cease after a few minutes, so that we were afraid to stop the artificial respiration. With the day came hope; the attacks of opisthotonos were less frequent and severe and the patient would breathe for a considerable time without help. Between 7 A.M. and 1 P.M. artificial respiration was occasionally necessary, although the baby was frequently cyanosed.

So far the only stimulant and medicine given had been whiskey and chloral hypodermically, so great had been our fear of exciting absolute and final arrest of breathing if we introduced liquids into the mouth. At 1 P.M. the baby, being in a pitiable condition of exhaustion and malnutrition from lack of food and continual handling, we decided to make a desperate attempt to feed it by gavage. A soft catheter was pushed into the esophagus and two drams of breast milk injected into the stomach; no convulsion or stoppage of breathing occurred, possibly because the excitability of the pharyngeal reflex had

diminished—perhaps because the sensibility of the glosso-pharyngeal nerve was obtunded by the passage of the tube, and also because the food being injected into the stomach the highly complex muscular and nervous mechanism of deglutition was not employed.

The success of the procedure was complete. The baby was invigorated by the breast milk, and chloral was again given at 3 P.M. through the tube. The malady seemed to vanish. The baby, much revived, nursed its mother at 5 P.M. The infant was given chloral and carefully watched through the night; no recurrence of its symptoms took place.

The next day, the baby, although languid and apathetic, nursed well and afterward made a rapid and complete recovery.

The duration of the illness was from twenty-four to twenty-six hours and ran its course without fever. The excessively acute symptoms when life was sustained by artificial respiration and oxygen lasted nineteen hours.

CASE II.—Burckhalter. The boy was born after a normal labor and was in vigorous health up to the morning of the seventh day of life, when he nursed but little and seemed restless and vomited once. At noon the nurse found the baby cyanosed and breathing badly; almost immediately it stiffened backward in the attitude of opisthotonos and stopped breathing. By means of artificial respiration in a warm bath the child was made to breathe again. Nevertheless for the next two hours the baby had recurring attacks of opisthotonos, retraction of the head and trunk with shallow, irregular breathing.

I found the accoucheur making artificial respiration on a very rigid baby. After the spasm relaxed the infant was thoroughly examined. The cord was off, the umbilical wound was healthy, the child was apathetic, there was general muscular relaxation, the knee-jerks were very active; pupils dilated with no response to light; normal temperature; pulse strong and regular; respiration 30; heart and lungs, nothing found; arms and legs easily moved. During the remainder of the infant's life the attacks of opisthotonos returned every fifteen to twenty minutes.

The first symptoms were first irregular, followed by slow respiration, then an absolute arrest of breathing, the child being bent backward, the arms and legs were not stiffened, there was no trismus or twitching of the face. In fact, the principal symptom of the malady was the sudden cessation of breathing every quarter of an hour. So long did these periods of apnea last

that we were afraid to allow them to terminate spontaneously. The pulse for the first few moments of the spasm would be strong, then with the cessation of the breathing it would become irregular and weak. After witnessing several of these attacks, it became evident that the only way to keep the child alive was to make artificial respiration most of the time. Occasionally even this was unsuccessful and we resorted with some effect to mouth to mouth insufflation. To prevent the child from losing heat the room was kept very hot and the child lay in a warm bath. When natural respiration ceased, the baby was pale, only once did it become livid. Sometimes in the attacks there was frothing at the mouth, and nose bleeding. When breathing was for awhile reestablished, the rate was 30 to 36 to the minute; pulse 160 to 180; at 3 P.M. rectal temperature was 101°; between 6 and 8 P.M. oxygen was continuously given; it always had the effect of strengthening the heart. (I have since regretted it was not given longer.)

From hour to hour the infant grew weaker. Twice we attempted to give nourishment by the mouth; each time that milk or whiskey was poured into the mouth the baby would stop breathing, and so long did it remain with no effort at respiration that we did not venture to feed it again. At 2.30 A.M. the child died; no autopsy was permitted.

We were at a loss to explain the cause of the symptoms. Both meningeal-hemorrhage and tetanus neonatorum were suggested as conditions which are accompanied by respiratory disturbance and eclampsia.

DR. KEITH'S CASE.—He reports the occurrence of similar acute respiratory failure in a child eight days old. The infant had just been circumcised, had lost an unusual quantity of blood and had taken the anesthetic badly. He suffered from the same repeated stoppage of respiration and cyanosis and was apparently rescued from impending death by oxygen inhalations and artificial respiration. Dr. Keith's patient had no convulsions and the act of deglutition did not cause an arrest of breathing. One is in doubt from Dr. Keith's history as to the condition of the pharyngeal reflex. He says there was no attempt to swallow, and it was only possible to moisten the lips with brandy and milk.

The history of the case is about as follows:

August 27th the infant was circumcised.

August 28th at 3.15 A.M. the breathing was labored and the

face cyanosed; suddenly the respiration stopped and the child was apparently dead. Dr. Keith found the heart still beating, and revived the child by artificial respiration. When natural breathing was established he left the room and in a few minutes was sent for again, to find the child apparently dead again. Artificial respiration was made, and after twenty minutes natural breathing was established, only to cease again in a few minutes. The heart was very rapid and the color very bad. After this Dr. Keith made artificial respiration hour after hour. After 12.30 mid-day, oxygen inhalations were continuously used with great benefit; after fourteen and a half hours artificial respiration; the child was able to breathe entirely by its own effort; the oxygen was kept continuously playing into the child's mouth for twelve hours longer; was stopped for one and a half hours, when the baby grew livid and the oxygen was again used.

August 29th—Three A.M., temperature taken, 103.6°.

August 30th—Child feverish, with offensive stools; oxygen still continuously used; weak heart; child looked badly; Cheyne-Stokes respiration.

August 31st—One A.M., temperature 105°; tepid sponge at 1 and 4 A.M.; 6 P.M., temperature 101°; oxygen was stopped, as it was suggested that it might be the cause of the fever.

September 1st—Several attacks of cyanosis relieved by oxygen; 4.30 A.M., baby stopped breathing; brought to by artificial respiration; after this the child did well and had no more alarming symptoms.

Neither Dr. Keith nor his friends were able to give a reasonable explanation for the long cessation of breathing.

I must also confess my inability to classify my own two cases with any disease of the newly born with which I am familiar.

The three children whose history I have given all had symptoms closely resembling each other. In fact, I believe that they all suffered from the same malady.

In Dr. Keith's case the illness began on the eighth day of life. In my first case respiration failed on the fourth day. In my second, respiration failed on the seventh day. None of the children were born after a difficult labor; none were asphyxiated, had trismus, or had an infected umbilicus. Dr. Keith does not allude to any infection of the umbilicus or the circumcision wound.

All the children suddenly ceased breathing without any assignable cause. This respiratory failure recurred again and again, so that Dr. Keith and I feared that life would shortly become extinct, unless artificial respiration was continuously made. None of the children had laryngismus, as air passed easily into the larynx. Both of my cases had frequent attacks of opisthotonos often associated with the respiratory failure. My cases ran their course practically without fever. Dr. Keith's case developed fever 102° to 105°, on the third and fourth day of its illness.

DURATION OF SYMPTOMS.—Burckhalter: This baby died after the apnea and opisthotonos had lasted fourteen and a half hours.

D. W.—In this patient the acute failure of respiration and opisthotonos subsided after twenty-six hours; the most acute stage was nineteen hours.

Dr. Keith's case: The respiratory failure and cyanosis were very severe for fourteen and a half hours; superficial breathing, cyanosis with fever continued for about seventy-two hours longer.

It seems to me that the three cases with their sudden failure of respiration were afflicted with a rare and unnamed disease of the newly born. Possibly we contended with some transitory but powerful infection of the medulla upon which the toxins played for a few hours with terrific force. The cessation of breathing was due, I believe, to some powerful inhibitory stimulus upon the respiratory centers in the medulla. This bulbar theory of the malady is further strengthened by the existence of morbidly active pharyngeal reflexes, that is, the contact of liquids in the pharynx stopped breathing. It will be recalled that excitation of the glossopharyngeal nerve will cause in health a short arrest of breathing. Both the pneumogastric and the glossopharyngeal nerve have practically the same nucleus in the medulla. Thus any morbific influence acting upon the origin of the vagus would also impair the function of the glossopharyngeal nerve.

Although disturbances of breathing and opisthotonos would suggest tetanus neonatorum, yet the condition lacked the essential symptom of trismus; there was no rigidity of the jaw; no contraction of the masseters.

Convulsions of the newly born are sometimes due to the cerebral injury or meningeal hemorrhage. None of the patients

had a history of an injury to the head, and the two that survived showed no paralysis or permanent lesion of the nervous system.

As all general infections of the newly born may cause a noticeable disturbance of the rhythm of breathing it is possible I have described as a disease a symptom which may occur in any malady of early life—as, umbilical septicemia, tetanus or pneumonia.

Dr. Thompson S. Westcott records (ARCHIVES OF PEDIATRICS, October, 1897) a similar condition occurring in a four-weeks-old child suffering, with several other members of the same family, from influenza. The baby had been ill four and a half days with grippe; suddenly it became cyanotic and breathed very superficially. It was revived by heat and stimulation. A second attack of cyanosis occurred five hours later, and a third attack was excited by swallowing milk. Evidently the same increased excitability of the glossopharyngeal nerve (observed in my two cases), existed in this child. Dr. Westcott states that the attacks would develop after a period of comfortable breathing. The respiration would become shallow with increasing cyanosis. Later an absolute stoppage of breathing took place—the thorax was motionless, the limbs rigid. These attacks continued for hours with great severity and frequency, and Dr. Westcott records twenty-nine periods of more or less dangerous apnea. He seems to have worked heroically with oxygen and artificial respiration, expecting at each seizure the child would die. Gradually the attacks became fewer and less severe. After a lull of some hours the child stopped breathing again, assuming the posture of opisthotonos. After this the patient improved, was occasionally cyanotic, and later had some bronchial engorgement of short duration. The disease ended with an alternating internal strabismus continuing several weeks.

The writer believes that the attacks of apnea were due to the pressure on the medulla, the rapid clearing up pointing to congestion or slight edema. This respiratory failure exactly resembles the apnea in the three cases I have narrated, which were uncomplicated by influenza. It is, of course, possible that this same sudden failure of respiration may be produced by several forms of infection, all affecting the highly sensitive respiratory centers of early infancy.

THE FEEDING OF AN INCUBATOR BABY.

BY CHARLES W. TOWNSEND, M.D.,

Boston.

S., a female, weighed at birth two pounds and twelve ounces, and was seventeen inches long. Considerable interest attaches to this light weight, owing to the fact that the infant was only two weeks premature, and the probable explanation lies in the fact that the placenta was found to contain numerous large and small areas of necrosis.

Dr. J. W. Williams, in his article in the *Festschrift* to Prof. Welch says "Marked infarct formation (in the placenta) is not infrequently observed and often results in the death or imperfect development of the fetus. It is usually associated with albuminuria on the part of the mother." This association existed in the present case.

The infant was wrapped in a wadded cotton and cheese cloth jacket and placed in an incubator, where the temperature was constantly maintained at 85° F., and where good ventilation was secured. The incubator, resembling that of Tarnier, consisted of a box with a glass lid through which the infant could be seen lying on a shelf. Below the infant a tank refilled at regular intervals with hot water maintained the requisite temperature of 85° F. with a variation of only two or three degrees as shown by a thermometer within the lid. Holes at the side near the bottom for the entrance and an anemometer at the other end on top for the exit of air served for ventilation. The constant revolutions of the wheel of the anemometer showed that the ventilation was satisfactory. Here she remained constantly for six weeks, and was finally weaned entirely from this abode at six and a half weeks in warm weather.

The gain of weight was from the first most satisfactory. From a birth weight of two pounds twelve ounces, the infant doubled its weight in twelve weeks, tripled it in four and a half months, and more than quadrupled it at six months, weighing then eleven pounds and three ounces. At a year she

weighs seventeen pounds, or more than six times her birth weight.

During the first six months the infant averaged a gain of five ounces a week, the least gain being one ounce for one week only, the greatest gain eleven ounces. The length at birth was seventeen inches, at six months, twenty-three inches, or an average gain of an inch a month.

The infant was fed at first from a Breck premature feeder, which is simply a large dropper holding an ounce, with a small nipple at the end.

The food from the first was cow's milk modified at home by a method that I have used for some years, and have found most simple and satisfactory in practice even in difficult cases. The results in this infant were certainly good, and although I have no intention of using this one case as an argument for this method of feeding, it will serve as an illustration, and at least shows that it is possible to successfully feed a very small infant on a simple home mixture.

There is nothing new about the method of feeding, except that it provides a way for easily calculating the approximate percentages of fat, sugar and albuminoids in the mixture, and although these calculations are only approximate, they are, I believe, sufficiently accurate for practical purposes. This knowledge of the percentages of the food is a great help in the feeding problem.

The rule I have worked out is this: Each ounce of 10 per cent. cream in a 20-ounce mixture represents .50 per cent. of fat, .20 per cent. of sugar and .20 per cent. of albuminoids; and each even tablespoonful of sugar of milk added to this mixture raises the percentage of sugar 2.*

Cream containing 10 per cent. of fat is obtained, not by the centrifugal process, but by pouring off the upper quarter from a bottle or can of milk after it has stood five hours. This, of course, is approximate, but I have found that the amount of variation in the cream obtained in this way is not very great, much less than the variations in commercial creams. Centrifugal creams are, in my opinion, undesirable.

The advantage of raw over sterilized or even pasteurized milk is in many cases so great that I prefer the uncooked milk, if

* See C. W. Townsend, *Boston Medical and Surgical Journal*, March 23, 1899, and October 11, 1900.

it is a possible thing. With perfectly fresh and clean milk in the present case, it was always used raw.

A very weak mixture was given the infant at the first as follows: Top milk, 1 ounce; water, 18 ounces; lime water, 1 ounce; sugar of milk, 2 tablespoonfuls, or approximately, fat, .50; sugar, 4.20; albuminoids, .20. A drachm was given every hour during the day, two drachms every two hours during the night.

In difficult cases, where the power to digest the caseinogen is slight, I often increase the amount of albuminoids in these weak mixtures by the addition of the whites of one or two eggs in the twenty ounces. The white of egg resembles lact-albumin closely and is so much easier to obtain than whey, it seems to me it is better and safer in milk modifications.

The strength of this mixture was gradually increased by adding one ounce more of top milk, and one ounce less of water until on the seventh day it was as follows: Top milk, 5 ounces; water, 14 ounces; lime water, 1 ounce; sugar of milk, 2 tablespoonfuls, or fat, 2.50; sugar, 5; albuminoids, 1. In some cases it is necessary to increase the strength more slowly by half ounces or even quarter ounces daily, an increase of only .25 per cent., or .13 per cent. of fat, and .10 per cent. or .5 per cent. of albuminoids respectively.

The infant was now taking three to four drachms every hour and a half in the day and every two hours at night. At two weeks the strength had reached: fat, 3; sugar, 5.20; albuminoids, 1.20, and the infant was taking five drachms every hour and a half. This was continued until the end of the first month when the strength was increased to: fat, 3.50; sugar, 5.40; albuminoids, 1.40, and fourteen drachms were taken at a feeding.

At three months the food was ordered as follows: Top milk, 8 ounces; water, 11 ounces; lime water, 1 ounce; sugar of milk, 2 tablespoonfuls, or approximately, fat, 4; sugar 5.60; albuminoids, 1.60. About the average strength of woman's milk.

The infant's progress was practically uninterrupted. She cut her first tooth when six months and one week old.

The only other points I wish to speak of in connection with the case is as regards the use of Jersey milk and the addition of cereals. When the baby was three months old it

seemed necessary, in moving to the country, to use Jersey milk, and it was hoped this would be digested, as the analysis of one specimen showed only 3.85 per cent. of fat. Although the baby gained regularly on this Jersey milk an average of four ounces a week, the dejections were increased in number although generally well digested, but there was considerable wind. The milk was therefore changed to the mixed milk of Holstein and Ayrshire cows with immediate improvement in the digestion, and an increase of weekly gain in weight to ten ounces the first week, and eight ounces for each of three succeeding weeks.

From the eighth month on a cereal in the form of either barley or oatmeal water was added to the diet. Had she fallen off either in appetite, digestion or gain of weight I should have begun on a cereal before that time. I believe that most infants do better by the addition of barley or oatmeal water as a modifier when they reach the age of six to eight months, but that although the majority do not need the cereal before this age, there are some cases that do need it and do much better with it.

I have recently fed in a similar way a baby born at the seventh month or thirty-second week. Here the gain weight, after the initial loss of five ounces, averaged six ounces a week for the first three months, the birth weight of five pounds and one ounce being a little more than doubled in that time.

In conclusion I would make a plea for simplicity in infant feeding, with at the same time approximate accuracy in percentages.

DISCUSSION.

DR. CHAPIN.—I would make one criticism on the paper and that is that I do not think it possible to get 10 per cent. of cream uniformly by the method the author pursues. I have had a great many assays made and I find that when using ordinary milk with a 4 per cent. butter fat that if the first twelve ounces be taken off with a dipper from the top I get uniformly a 12 per cent. cream.

DR. MORSE.—Speaking of feeding premature babies I think it is possible to bring them up on modified milk but I do not think that is the best way to feed them. It is even more important for premature babies than for babies born at full term that they should be fed on natural breast milk.

DR. ROTCH.—I think that breast milk is preferable when you can get it and when the child can take it, but it often happens that you cannot get a breast milk to suit the particular baby. It is hard enough to make the milk of a wet nurse suit any baby, and it is still more difficult to suit the premature baby.

DR. TOWNSEND.—In answer to Dr. Chapin I would say that I have not had a great many examinations made and that in pouring off the milk we get a certain amount of the lower milk which reduces the percentage below 12 and the average is about 10. I agree with Dr. Morse that breast milk is preferable if it can be obtained, but I have found that in many cases of premature feeding the mother dislikes the action of the breast pump and this results in a changed character of the milk obtained. The percentages I gave are approximate only. Of course we cannot have the milk analyzed in every case. A certain amount of clinical experimenting is necessary, as is also the case even if we have the exact analysis.

AN ANALYSIS OF THIRTY-TWO CASES OF CONGENITAL HEART DISEASE.

BY JOHN LOVETT MORSE, A.M., M.D.,

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During the past few years I have seen thirty-two cases of congenital heart disease on which I have kept careful notes. In the hope of learning something as to the prognosis of one of them from the study of the others, I recently looked them all up. The analysis of the results, although showing nothing of importance, offers some points of interest.

CLASSIFICATION.—Although the extreme difficulty, and in many cases the impossibility, of making an accurate diagnosis of the pathological condition during life was recognized, nevertheless, an attempt was made to classify the cases in a general way. The diagnosis of patent foramen ovale was made in 6 and a defect in the ventricular septum seemed most probable in 6 others. In 17 the chief lesion was thought to be at or about the pulmonary orifice. In 5 of these patency of the ductus arteriosus seemed probable. In 3 not even a guess as to the situation of the lesion seemed justified.

ETIOLOGY.—Fifteen of the cases were in males and 17 in females, all classes, moreover, being nearly equally divided between the sexes. Two of the cases with patent foramen ovale were in premature infants. All of the others were born at full term. Prematurity seems, therefore, to be a more important factor in the etiology of patent foramen ovale than of other conditions. Other malformations were uncommon. One case had two club feet and a club hand. One was microcephalic and another developed hydrocephalus when about a year and one-half old. All of these were in cases in which the lesion was located in the region of the pulmonary orifice.

ONSET.—In 14, or 44 per cent., the cardiac condition was discovered during a routine physical examination, there having

been no symptoms pointing toward the heart and the cardiac disease being unsuspected. The diagnosis of the cardiac condition in these cases was patent foramen ovale in 1—in a total of 6; defect in septum in 2—in a total of 6; lesion at the pulmonary orifice in 10—in a total of 17; unclassified in 1—in a total of 3. The age at which the cardiac condition was discovered in these cases varied from six weeks to two and one-half years, the average being about ten and one-half months. In 4 of these no symptoms had developed at the end of two, three and one-quarter, three and one-half and four years. The symptoms for which the others were brought were cyanosis, 17; dyspnea, 6; cough, 5; convulsions, 2; edema, 1. One was brought especially on account of peculiar abdominal respiration and another for "backwardness." The earliest symptom noted was cyanosis in 11, dyspnea in 1 and cough in 1. In the others several were noted together.

CYANOSIS.—Cyanosis was, as already stated, the most common symptom. It was present at birth in 7, 3 of which were supposed to be cases of defective ventricular septum. It developed in 12 from nine days to three years after birth; in 6 it developed in the first two months, but in 2 not until three years, in 1 of these following pertussis. In 6 cases which died it did not develop at all or only just before death. Eight who are alive at the ages of two months, seven months, two years, two years, three and one-quarter years, three and one-half years, four years and seven years, have never shown it. In 4 the cyanosis disappeared after four days, fifteen days, one year and one and one-half years.

OTHER SYMPTOMS.—Analysis of the other symptoms is impossible. Several had convulsions from time to time, which were evidently the result of cyanosis, while others had recurrent attacks of severe dyspnea without apparent exciting cause. Dyspnea was common in the older children and a number were subject to "bronchitis" and cough. Edema occurred but once. The development of 5 of the 6 cases of supposed open foramen ovale was normal at the time of writing. That of 8 of 12 of the other living cases was far below normal, several of them being very small and wizened.

PHYSICAL SIGNS.—In 2 of the cases diagnosed as patent foramen ovale nothing abnormal was detected in the heart. In

the 4 other cases classified as patent foramen ovale murmurs were present, but no cardiac enlargement or other abnormal physical signs were made out. In the 6 cases in which the lesion was supposed to be located in the ventricular septum, the cardiac area was increased in 5, the enlargement in all cases being in both directions, more to the left in 2, more to the right in 3. It developed in the first few months in all. Of the 17 cases of supposed disease at the pulmonary orifice it was increased in 7, the enlargement being almost entirely to the right and developing early. The area was normal in the 3 unclassified cases. That is, the area was increased in 12 of 26 cases in which the lesion was probably not a patent foramen ovale. The size of the heart diminished decidedly under treatment in 1 case, and in 2 others the murmur disappeared. A thrill was present in only 2 cases. The extremities were clubbed in but 6. The spleen was enlarged in 7 and the liver in 3 cases. Clubbing of the extremities and enlargement of the liver and spleen were common in the cases in which the lesion was located at the pulmonary orifice.

MORTALITY.—Five of the cases were lost sight of at the ages of one and one-half months, three and one-half months, seven months, eighteen months and two years. None of the cases diagnosed as patent foramen ovale have died. Five are living at the ages of seven months, three years, three and one-half years, four years and six and one-quarter years. Twelve of the other cases are dead. Six died of the heart disease at three, six, eight, nine, ten and twelve months; 6 of other diseases at four, six, eight, nine, nine, and twenty-four months; 10 are alive at the ages of two, and twenty months, two, two and one-half, two and three-quarters, three and one-quarter, four, five, seven and seven years.

Five of the cases have passed through measles; 2, scarlet fever, and 2, whooping-cough. None have died of contagious diseases.

CONDITION OF LIVING CASES.—George G., two and three-quarter years (pulmonary orifice). Hydrocephalic; idiotic; cyanotic; small; extremities clubbed; liver and spleen enlarged.

Margaret H., two and one-half years (unclassified). Small; cyanotic. Can walk but little because of dyspnea.

Frank McC., five years (pulmonary orifice). Small; cya-

notic. Subject to "colds." Unable to play much because of dyspnea.

Mary L., seven years (defect in septum). Small; slightly cyanotic. Goes to school. "Well." Runs and plays but gets out of breath quicker than other children. Subject to "colds."

Gertie B., seven years (defect in septum). Small; no cyanosis. Goes to school. Plays a little. Subject to "bronchitis" and dyspnea.

Louis S., three and one-quarter years (pulmonary orifice). Small; rachitic; very backward. General condition improved much in past year and size of heart much diminished. No cyanosis; no symptoms.

Esther M., two years (pulmonary orifice). Small; backward. No symptoms.

Charles D., seven months (open foramen ovale). Rather small; slight cyanosis of face. Improving. Murmur intermittent.

Mary B., two months (pulmonary orifice). Normal. No symptoms.

Harry G., four years (pulmonary orifice). Normal. No symptoms.

Dorothy B., three and one-half years (open foramen ovale). Normal. No symptoms.

In these cases the cardiac condition continued unchanged, compensation being more or less complete. In the following cases, however, recovery apparently took place:

Esther W. was first seen March 25, 1899, when four weeks old. The cardiac condition was discovered during the routine examination. On questioning, the mother said that the child had always been blue. The first sound over the whole precordia was replaced by a very high-pitched, blowing murmur which was transmitted both upward and into the axilla. There was no visible impulse. The second sound was everywhere distinct. The cardiac area was not increased. There was no cyanosis when quiet but it was marked on crying. The physical examination was otherwise negative. She was seen again November 23, 1900, when twenty-one months old. The mother said that the cyanosis on crying continued for some months and that she used to have fainting fits. For some months, however, she had been perfectly well and was able to run and play without dyspnea or discomfort. The area of the heart was

normal. The sounds were perfectly normal in every way. There was no cyanosis even when she cried vigorously.

Lena R., was born August 20, 1894. after a perfectly normal labor. She was healthy at birth. When a small baby she had trouble in breathing when she was held upright. In January, 1895, she had a "fit." She then began to have more trouble with her breathing and attacks of blueness. The condition of the heart was then discovered by the attending physician, Dr. Howe, of Newburyport. The dyspnea and cyanosis increased progressively from this time. I first saw her December 28, 1895, when she was sixteen months old. She was well developed and nourished. The anterior fontanelle was not quite closed. She had four teeth. There was a slight rosary. She was able to sit up but could not stand. Dyspnea was marked and cyanosis was marked and general. The cardiac area was normal. The impulse was in the sixth space in the anterior axillary line. There was a suspicion of a thrill at the apex. At the mitral area the first sound was roughened and continued into a short, harsh, blowing murmur. The second sound was distinct. The murmur was audible over the whole heart except at the pulmonic area. The second pulmonic sound was accentuated. The lungs were full of dry râles. There was no enlargement of the liver or spleen. The extremities were not clubbed.

In a letter dated January 16, 1901, when she was six and one-half years old, Dr. Howe states that she has had no cardiac symptoms since she was two years old. She has survived both measles and scarlet fever and is now a plump, active little girl. The heart action is somewhat irregular, but otherwise normal.

When these cases were first seen no doubt whatever was entertained as to the accuracy of the diagnosis of congenital heart disease. The second case seemed an especially severe one and an early demise was expected. In spite of the unlooked-for recovery there can be but little doubt, I think, that there really was an organic lesion in these cases. It is possible that the recovery is apparent rather than real and that the cardiac lesion still persists, the absence of symptoms and physical signs being due to an unusual amount of compensation. There seems no way of settling these questions without autopsies. Even then, however, positive answers might not be obtained, as the presence of normal conditions would not show whether or not abnormal conditions had previously existed.

John T. was born May 29, 1898, after a difficult instrumental labor. He was a full-term baby and normal in every way. He was moderately jaundiced for two days. On June 9th, when he was asleep, his mother noticed marked general cyanosis. There were no other symptoms. The doctor, who arrived soon after, found the temperature 97.2°F., but noted nothing abnormal about the heart. I saw him twenty-four hours later.

He was then but moderately cyanosed and seemed comfortable in every way. He was small and fairly nourished. The area of the heart was normal, the sounds were normal, the temperature was normal. Physical examination was otherwise normal. The diagnosis of a congenital heart lesion was made and the prognosis of that condition given.

He improved rapidly in color, the cyanosis disappearing in the course of a couple of weeks. Since that time he has been absolutely well and has had no recurrence of cyanosis or any other symptoms of heart disease. The physical examination of the heart has remained normal.

Mary C., was born May 17, 1897, after a normal labor. She was a full-term baby and normal at birth. She apparently did well until the ninth day when blueness was noted at times about the mouth as well as coldness of the extremities. This slight cyanosis and coldness of the extremities continued. On the nineteenth day, after working hard at the breast, she became blue, then pale, and ceased to breathe. With artificial respiration and stimulation she began to breathe again and gradually improved. At this time the area of the heart was normal and there were no murmurs. There was general, moderate cyanosis. The physical examination was otherwise negative. The cyanosis diminished gradually and was all gone in about ten days.

She has developed normally, has had no return of the cyanosis, and at present shows no abnormal physical signs.

In these cases also no doubt was felt at the time, either by myself or by the physicians with whom I saw them, as to the presence of an organic heart lesion. In both cases the condition was thought to be a patent foramen ovale. The rapid recovery in these cases is not inconsistent with that diagnosis, as the foramen may close even after considerable delay. This is probably what happened. It is not impossible, however, although

decidedly improbable, that the collapse and cyanosis may have been due to some other cause or causes.

The points brought out by the study of these cases which seem to me of most interest are:

1. The considerable proportion of cases in which the cardiac lesion was discovered during a routine physical examination, there having been no symptoms referable to the heart.

2. The length of time which the condition may exist without the development of any symptoms, several cases having shown no symptoms at three and four years of age.

3. The comparative mildness of the symptoms in cases of patent foramen ovale.

4. The recovery from lesions which from physical examination were apparently the same as those in cases which resulted in chronic invalidism or death.

70 BAY STATE ROAD.

APPENDICITIS IN CHILDREN OF TWO YEARS AND UNDER.

BY J. P. CROZER GRIFFITH, M.D.,

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The following case of appendicitis occurring in an infant seems worthy of being placed on record:

Louise Budd, aged three months, colored, entered the Children's Hospital, Philadelphia, April 13, 1901. She was a well-nourished child, of whom practically no earlier history could be obtained previous to April 9th, on which date she began to suffer from diarrhea, with mucus in the stools. This continued until the next day, when fecal movements ceased, although some blood is said to have been passed upon the evening of this date. Vomiting began about the same time. Nothing at all had passed from the bowels since the 10th of the month.

On examination at the hospital, at 6 P.M. on the evening of admission, the baby was found to be clearly very ill, in a state of collapse, with a bad color, rapid and weak pulse, sunken eyes, and with the abdomen so distended with gas that palpation was unsatisfactory. No stools occurred and there were no straining efforts. A rectal examination gave negative results. With the idea that intestinal obstruction might be present, large enemata were ordered, but only a very small quantity of mucus with reddish-stained fecal matter was obtained.

The infant revived only very slightly by 10.30 P.M., and operative interference was felt to be beyond consideration. A slight amount of greenish material was vomited at 7.30 A.M. on April 14th, and the baby died a few minutes later. The temperature while in the hospital ranged from 102 to 105.5° F., generally being over 104°. The notes of the lesions formed at the autopsy furnished me by Dr. Alfred Hand, Jr., the pathologist of the hospital, read as follows:

"Considerable offensive, turbid, yellowish fluid is present in the peritoneal cavity, and scattered flakes of lymph, easily

removable, are seen on the parietal and visceral layers of the peritoneum. The appendix points to the left and is about 6 cm. long. The distal half is freely movable and of a blackish-red color which extends to a point of constriction at the middle of the appendix. Just beyond this constriction, towards the tip of the appendix, the organ is crossed by a band of lymph, which is attached at one end to the mesentery of the ileum, and at the other end to the peritoneal coat of the ileum. This would naturally be thought to be the cause of the gangrene were it not that the appendix is gangrenous for a distance of 2 mm. above the band, *i.e.*, towards the cecum, and that the band is not tightly adherent to the appendix.

"On releasing the appendix from the adhesion, the mesentery is found to extend practically only half way to the tip, although there is a narrow prolongation of it nearly to the tip. On both sides of the mesentery, along the proximal half of the appendix, are two bands of connective tissue like guys or accessory mesenteries, which render the proximal half of the appendix comparatively immovable while the distal half is freely movable.

"For microscopic examination the appendix was dissected into three pieces. Transverse sections were cut of the tip and of the cecal end, and longitudinal sections of the central constricted part.

"The cecal end is normal, showing tubules of healthy mucus membrane, surrounded by the normal masses of lymphoid cells. This is also seen in the longitudinal section, but as the constriction is approached, connective tissue increases to a large amount and the mucosa abruptly disappears at the constriction. In the distal part there are two processes visible, an acute and a chronic. The chronic inflammation is evidenced by an increase of connective tissue and the presence of organized blood-vessels; the acute by tremendous congestion, extravasation of blood, and a complete degeneration of the epithelial cells, a granular detritus remaining to show the former location of the tubules.

"The adhesion on section showed an aggregation of round cells, with a tendency to the formation of new connective tissue at one edge. This band was evidently of very recent formation."

The cause of the disease in this case, as far as this can be

determined, appears to have been a kinking of the appendix, due probably to the short mesentery and to the other unusual and unusually tight bands which kept the lower half of the appendix firmly fixed while the distal half was free to move. There were no concretions or foreign bodies present in the appendix, and nothing else discoverable to account for the obstruction to the lumen and to the circulation which had evidently existed in the terminal portion of the organ. Both Monti (*Kinderheilkunde*, Bd. I., p. 497) and Sonnenburg (*Verhandlungen* d. XIII., *Cong. f. inner Med.*, p. 262) have called attention to a special tendency to kinking of the appendix present in children, and that inflammation of the appendix results from any cause obstructing its lumen is well known.

No lymph whatever was deposited upon the almost gangrenous distal portion of the appendix. The band of lymph described as crossing the middle of the organ was clearly not the cause of the constriction, as the narrowed portion was situated slightly nearer the cecum, as has been pointed out in the pathologist's report. It is also to be observed that this infant had clearly at some previous time suffered from an attack of appendicitis, more or less severe, as is indicated by the chronic lesions discovered.

Appendicitis may occur at any age, but is commonest in adolescence or in early adult life. I have myself been fortunate in seeing two cases in children of four and four and a half years respectively which I have reported elsewhere (ARCHIVES OF PEDIATRICS, August, 1898). Each of these was operated upon and pus found, but in neither could the appendix be discovered. Recently I have had the opportunity of seeing a case in a child of three years operated on in the Methodist Hospital in the service of Dr. Spellissy. Here, too, an abscess cavity was evacuated but the appendix was undiscovered.

The relative frequency of appendicitis at different ages in children may be seen from the following statistics: Brun (*Traité des Mal. de l'Enf.-Grancher.*, III., p. 106) in 45 cases in children observed by himself reports 3 from one to five years of age; 20 from five to ten years, and 22 from ten to fifteen years. Fitz (*Transac. Assoc. Amer. Phys.*, 1886, p. 106) in 228 cases of appendicitis at all ages collected by him, found 22 from twenty months to ten years and 86 from ten to twenty years. Matterstock (*Gerhard's Handb. f. Kinderkr.*, IV., 2, p. 899) in 72 col-

lected cases in children found 2 under two years (1 of seven months and 1 of twenty months), 10 from two to five years, 25 from five to ten years, and 35 from ten to fifteen years. Bamberger (quoted by Matterstock) in 73 cases in children found 2 less than two years of age and 20 from fifteen to twenty years. (I have been unable to verify the references given for this statement.) Gordon (*Thèse de Paris*, December 23, 1896) in 79 collected cases in children found 5 from two to five years, 33 from five to ten years, 35 from ten to fifteen years. Only one of the first five cases was not over two years old. Fenwick (*Lancet*, 1884, II., p. 987) in 97 collected cases, irrespective of age, found 9 below the age of ten years, half of these being under five years. Jalaguier (*Traité de Chirurgie*, IV., p. 637) in 182 personally observed cases found 4 from one to five years, 42 from five to ten years and 64 from ten to fifteen years. Barthez and Sannée (*Traité des Malad. des Enf.*, I. p. 473) have seen 23 cases in children, of which 5 were from four to seven years old. Hawkins (*Diseases of the Vermiform Appendix*, p. 62), out of 224 cases of all ages occurring in St. Thomas' Hospital, found 26 in children of from five to ten years. E. McGuire (*Virginia Medical Semi-Monthly*, 1898-9, III., p. 400), out of 104 collected cases in children, reports 3 under three years, 47 between the fourth and ninth years and 54 between the tenth and fourteenth years. Deaver (*New York Medical Journal*, 1893, LVIII., p. 342), says the youngest patient he has seen was a child of two years.

The cases in these statistics certainly duplicate each other to some extent. They show that the disease is decidedly uncommon under the age of five years, and especially so in infancy. At the age of two years or less the affection can certainly be called rare, and the younger the child the less common it appears to be. Medical literature shows very few cases indeed. The following list of the detailed cases of two years or less duplicates to a degree which cannot be determined the number included in the collective statistics given. I have arranged them according to the age, and have placed my own case in order in its proper position:

1. Pollard (*Lancet*, 1895, I., p. 1114). Boy of six weeks, hernia of the appendix into the scrotum; abscess; operation; no perforation; recovery.

2. Goyens (*Gaz. med Belge*, XII., No. 14; Abstract in ARCHIVES OF PEDIATRICS, March, 1900, p. 209). Boy of six weeks; perforated appendix discovered at autopsy.

3. Demme (XXIII. *med. Bericht. ü. d. Thätigkeit d. Jenner'schen Kindersp. in Berne*). Girl of seven weeks; appendix containing a concretion of porridge found at autopsy; no perforation.

4. Savage (*New York Medical Record*, April 23, 1898, p. 600). Boy sixty-one days old; hernia of appendix into scrotum; operation; appendix perforated; removal; death.

5. Griffith (case now reported). Girl three months; gangrenous appendix found at autopsy; general peritonitis; no perforation.

6. Elder (*Montreal Medical Journal*, March, 1901, p. 201). Boy of seven months; scrotal hernia of cecum and appendix, abscess; operation; appendix perforated; removal; recovery.

7. Betz (*Memorabilien*, 1870, XV., p. 118). Boy of seven months; perforated appendix found at autopsy. (This is the seven months' case included in Matterstock's list.)

8. Silbermann (quoted by Talamon "Appendicite et Perityphlite," 1892) saw a case of appendicitis in a child in the last period of lactation. (No reference to original found.)

9. Taylor (*Boston Medical and Surgical Journal*, May 20, 1897, p. 482). Boy of one year and three days; supposed to be intussusception; operation; perforated appendix found; death.

10. Monks (*Boston Medical and Surgical Journal*, June 5, 1890, p. 543). Boy of thirteen months; hernia of appendix into scrotum; abscess; operation; removal; no perforation; recovery.

11. Hauck (*Medical Review*, St. Louis, 1895, XXXI., p. 463). Girl of sixteen months; supposed to be intussusception; operation; appendix strangulated by coil of small intestine; no perforation; recovery.

12. Millon (*Arch. de Méd. Infant*, 1899, II., p. 285). Girl of nineteen months; appendicular abscess incised; appendix not found, but a stercoral concretion discharged later; recovery.

13. Holmes (*British-American Journal of Medical and Physical Science*, March, 1847, p. 285). Boy of twenty months; perforated appendix found at autopsy. (This is the twenty months case of Matterstock's list.)

14. Summers (*Philadelphia Medical News*, 1891, LIX., p. 513). Boy of twenty-two months; operation; appendix perforated; recovery.

15. Gordon (*Thèse de Paris*, December 23, 1896). Boy of two years; abscess; operation; appendix perforated; recovery.

It is also stated by Powers and by Jalaguier that a case in a child of six months has been reported by Tordeus. I believe this to be an error. The only reference I have been able to discover is to one of a child of six years (*Jour. de Med. de Chirurg. et de Pharm.*, Bruxelles, 1885, LXXX., p. 220).

Doubtless there are other cases published, but those quoted include all I have been able to find after a somewhat extended search.

It is interesting to note that in 9 of the 15 cases given above the appendix was found to be perforated; that in 4 cases the appendix had descended into the scrotum; that 9 cases were operated upon with 7 recoveries; and that in 2 cases the disease had been diagnosed as intussusception.

DISCUSSION.

DR. SHAW (guest).—The case has interested me very much because early in Jaunary, 1901, I saw such a case of appendicitis in an infant seven weeks old. The condition was not diagnosed during life but was determined at autopsy. The appendix was sloughed off and lay in the midst of pus. The infant weighed only six pounds, six ounces. There did not seem to be pain on palpation of the abdomen.

MATERNAL IMPRESSIONS—REPORT OF A CASE.

BY B. K. RACHFORD, M.D.,

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The mother of the child, whose history I here report, is gouty. She had had three or four attacks of slight catarrhal appendicitis each year for fourteen years. These attacks usually incapacitated her for one day, and the soreness in the region of the appendix entirely disappeared on the second day. The attacks were characterized by colic, and some pain and tenderness in the region of the appendix.

Thirty-six hours before the birth of her first child, in June, 1898, she had an attack more severe than usual accompanied by a fever of 102° F. Following the birth of this child there was a marked sepsis for twelve days, which could not be accounted for by anything in the pelvis, and which in the light of later events is believed to have been due to appendicitis.

In February, 1899, when she was two and a half months pregnant with her second child, she had one of these attacks, following which the enlarged appendix was clearly mapped out. Six days later the appendix was removed. The operation was quickly and skilfully performed, and the patient went off the operating table in excellent condition. Soon after her return to consciousness, however, she commenced to vomit, which symptom continued almost uninterruptedly for forty-eight hours. This attack of vomiting was thought to be due to the anesthetic. After the vomiting ceased everything went well until the tenth day when a stitch abscess developed on the outer side, and at the top of the wound. Although the stitches had been taken out on the seventh day, sepsis gradually extended through all of the stitch wounds, producing considerable sloughing on both sides of the scar marking the line of incision. The sepsis was confined to the region of the stitch wounds. After a time the mother convalesced satisfactorily, but there now marks the site of the operation two very pronounced scars, on either side of the incision scar.

On September 20th a girl baby was born, weighing nine pounds, which was pronounced by the obstetrician to be a normal and well-developed child.

I saw this child professionally for the first time in November, when she was two months old. During the next two or three months, she had more or less continuous fever of low grade and obscure origin. In the early part of this attack the liver was greatly enlarged.

Throughout the winter the child continued to be imperfectly nourished, but during the summer, which was spent in Michigan, she completely recovered. On her return to the city in September, 1900, when she was one year of age, the mother noticed a mark resembling a stitch-scar located over the appendix. During the next six months three more stitch-scars appeared, four in all, located exactly in the region corresponding with the mother's scar, produced by the appendicitis operation. These markings became more pronounced, so that at the present time the appearance is such as would be produced by an operation for appendicitis, except that there is no scar marking the line of incision. The stitch-scars are, however, so pronounced that they can be seen across the room.

It is to be especially noted that the scar, as it is reproduced in the infant, resembles the appendicitis scars of the mother only in the stitch-scars and not in the scars which resulted in the incision and the lateral sloughings. Moreover these markings, while distinct, are not made of true scar tissue.

It is my belief that the maternal impressions which produced the markings on the child resulted from the strain on the stitches in the abdominal wound of the mother during the forty-eight hours of vomiting, immediately following the operation.

It is worthy of note that the markings on the child were not noticed until the child was one year of age, and for the next six months they gradually increased in distinctness. During the past four months there has been no noticeable change. It is also noteworthy that the maternal impressions which produced the markings on the child occurred about one week prior to the termination of the third month of uterogestation.

In conclusion, I wish to say, that Dr. Arthur W. Johnstone, Dr. F. Forchheimer, and Dr. W. D. Porter, are almost as familiar with this child's medical history as I am. They have watched with me from time to time the development of these

markings, and are quite prepared to testify with me that the existing circumstances were such that we could not have been deceived in the essential facts as they are here reported.

DISCUSSION.

DR. WENTWORTH.—I would like to ask Dr. Rachford whether the marks on the child were in the nature of pigmentation or of scar tissue.

DR. RACHFORD.—They are neither pigmentation nor scar tissue but depressions in the skin.

MILK-SUGAR IN INFANT FEEDING.

BY A. JACOBI, M.D., LL.D.,

Professor of the Diseases of Children, Columbia University, New York.

The identity of milk-sugar in woman's and in cow's milk is very doubtful, and the milk-sugar of the market is quite often impure. That alone makes it desirable or advisable to substitute cane sugar if this afford the same advantage. These four dozen years I have made this very same substitution. Part of any kind of sugar (as also dextrin, peptone and salt solutions) is absorbed in the stomach. Most of the milk-sugar of the milk is changed into lactic acid by the bacterium lactis-aerogenes, bacillus acidi paralactici (not identical with Hueppe's bacterium acidi lactici, Y. Kozai, Z. Hyg., 31, p. 337) and perhaps twenty more different germs. Possibly, however, there is an additional source of lactic acid which appears during digestion from one-half to one hour after a meal, for it seems that this short time is not sufficient to give rise to such formidable masses of microbes as are required for the purposes of transformation. Possibly, therefore, there are several kinds of lactic acid, which differ not only in proportion to the temperature in which coagulation takes place and the composition of the nutrient soil of the bacteria. Still, all of this appears to be undecided.

Absorption of milk-sugar need not always be direct, however. Some of the milk-sugar which was not absorbed as such, is perhaps (Dastre) decomposed by microbic action or by hydrochloric acid, or by some unknown ferment, into galactose and glucose and then absorbed (Portier, in Soc. Biol., April 2, 1898). When eight-tenths of 1 per cent. of the milk-sugar contained in the whole milk in the stomach are changed into lactic acid, no more lactic acid is produced. Ordinarily this

limit is reached when about one-fourth of the milk-sugar has been changed into lactic acid. If at this period, however, lactic acid be neutralized by an alkali, then more milk-sugar is changed into lactic acid. In this way the amount of lactic acid present in the digestive tract and in its various derivata, depends on accidents only, that is, mainly on the presence or absence of an alkali, and it appears that in every preparation of cow's milk selected for the use of an infant there is milk-sugar enough to supply the needs of the digestive process. Moreover, a goodly part of the milk-sugar introduced, even in woman's milk, is eliminated unchanged, for Blauberg (Studien über Säuglingsfäces, p. 55) found the nursling's desiccated feces to contain from 0.22 to 0.59 per cent. of milk-sugar. Escherich found that peptones which form in milk are destroyed by acid fermentation, and concludes that another carbohydrate should rather take the place of milk-sugar in order to avoid the excess of lactic acid.

It appears after all this that it *is easier to give too much milk-sugar than too little,* and that the careful measuring and weighing of copious quantities of milk-sugar is of doubtful value, even if you know, or believe you know, that the milk-sugar you give and the milk-sugar of woman's milk are identical. Immediate fermentation in the intestine, moreover, should be carefully avoided for other known reasons. Lately Dr. Helen Baldwin has published (*Journal of Experimental Medicine,* Vol. V.) investigations which prove the formation of oxalic acid as the result of intestinal fermentation. It appears, therefore, that my method of adding to the cow's milk mixtures destined for infants and children, not milk-sugar but cane sugar, in moderate quantities estimated rather than anxiously weighed, was correct and justified by modern research.

Similar results were obtained by Rubner, Heubner and others, while lactic acid was noticed in the feces by Wegscheider, Uffelmann and Biedert. The milk-sugar is either absorbed or passes out in the feces, or (that is, the greater part of it) is changed into lactic acid. This should not be in excess.

What I have the honor of presenting to you in these words as read formed nearly literally a part of my report to the Children's Section of the Thirteenth International Medical Congress of 1900. The subject being of importance on account of its connection with infant feeding, than which there is no topic

apparently more simple but actually more discussed, contended and overwhelmed with uncertainties and real or imaginary difficulties, I bring the question of milk-sugar again before you with the intention of rather settling a single mooted problem than to open a discussion of all the topics connected with artificial infant feeding.

When I began the practice of adding cane sugar instead of milk-sugar to the artificial food of the infant, many of the theoretical reasons for so doing were not known, and many of the experiments made for the purpose of founding artificial feeding on a scientific basis were not yet performed. But what I have always asserted, even before some of my adversaries or some of the great experimenters of to-day consented to be born, is that observations on the living babies, particularly when repeated a thousand times, are at least of the same value as those made on test tubes. Though the physiological working of ingesta cannot be watched throughout the length of the alimentary canal, by means of any physical or intellectual Röntgen ray, their effects on the health and life of the infant may be noticed.

When these effects are proven to be salutary, it always takes more or less time for chemical and bacteriological research to confirm the results and to prove them to be self-evident. As an instance of this kind I remind you of the gradual change of opinions in regard to the administration of cereal (*i.e.*, in part amylaceous) decoctions in the artificial food of the very young. Clinicians like Heubner, Keller, or Gregor are converted to beliefs which some of them had long condemned; while physiologists and chemists are adding more and more actual proofs of the digestibility of amylum in the tract of the newly-born to the original observations of Schiffer, Zweifel and Korowin of nearly thirty years ago.*

In the same way my method of substituting cane-sugar for milk-sugar in infant feeding will find its way into practice; and, as has occurred before very frequently, the theories of the laboratory will follow the lead of a thousand practical experiences.

I can afford to wait—the babies must wait.

That is why I have been in no hurry to reply to a gently polemical footnote which my famous friend, Prof. Heubner, of

*Compare the experiments of E. Garte, *Gazetta Botkina*, Vol. xii., No. 1, on the action of the pancreas in the fetus and newly-born.

Berlin, publishes in connection with one of his very numerous but always interesting and learned articles on "Infant Feeding" (*Berlin Klin. Woch*, April 29, 1901). In his paper, "Die Energiebilanz des Säuglings," he speaks of the great differences of foods amongst races and peoples, of the increasing modern freedom ("Bewegungsfreiheit"), in regard to the nutrition of the weak and sick, which, according to my learned friend, "is almost not at all appreciated by pediatrists," though "Rubner has discovered the law of the isodynamic mutual substitution of nutrient substances." He points to the equal and salutary results which may be obtained in behalf of infants with different articles: Liebig's soup (old), Keller's malt soup, Heubner's and Hofmann's, and Soxhlet's and Heubner's milk dilution (very, very old), pure milk, buttermilk (not new), Gärtner's milk, and he might have added to his list considerably.

He might, for instance, have quoted the many proprietary articles tried by Biedert, that most learned, cautious, and successful but least aggressive and least pompous physician and author ("Dietetic Treatment of the Digestive Disorders of Children," 2d Ed., 1901). Even the teachings of Jacobi, which Hofmann ("Vorlesungen über Allgemeine Therapie," 1885, p. 224), declared to be most satisfactory thus far, might have come in for a passing notice.

According to my learned friend the possibility of nourishing a baby with results different from those of ten or twenty or thirty or two years ago is entirely due to Rubner's late writings. "When the young men will have read his modern works they will learn *wie hübsch es sich ausnimmt*," how pretty it looks "when Jacobi ventures upon giving instruction on the influence of the temperature of the atmosphere on decomposition and calorification in the organism." This influence I have acted upon and taught nearly half a century, and before Rubner was born, without claiming any priority. However, I do not believe that Rubner claims priority for the main principles underlying his researches, undertaken alone or in cöoperation with Heubner. Least of all shall I ever undertake to commit—that time has gone for me these fifty years—the crime of *lese-majesté* which consists in not ecstatically and vociferously recognizing the latest outcome of an author's experimentation or very newest magazine article, as new and *ne plus ultra*.

One more word: Dr. Heubner says that, my remarks quoted by him (piecemeal) from the *Archiv. für Kinderheilkunde*, Vol. XXXI., p. 17, leave him "absolutely cool." I suppose it is important that we should know that. I may be permitted to say that his remarks instead of leaving me "cool," warm me to the innermost of my heart. It is dozens of years that I have presented the principles extolled in Heubner's latest essay. It is true I never coined the term "balance of energy," or "quotient of energy." But I have been teaching the impropriety of limiting the feeding of infants to an unalterable formula, have refused to identify cow's and woman's milk, have denied the equivalence of the latter and cow's milk, no matter how prepared or how modified, have insisted upon the digestibility and usefulness of a certain amount of cereals, (amylacea), have asserted the beneficent latitude of nature as compared with the dictatorial utterances of our laboratory tyrants, of chemists and bacteriologists and other "authorities," have accumulated decade after decade of practical examples with my methods as first laid before European readers in the first volume of Gerhardt's "Handbuch," and taught to legions of American students and readers, have quietly stood by when I hardly ever was mentioned by the European workers in the same field, in connection with the subjects that appeared easily understood if approached in the spirit of cool, practical observation, and from the view of practical humanitarianism, and have finally found that what I practiced and taught has at last, though slowly and laboriously, in endless articles and self-styled discoveries, been recognized to be true. No less a man than Dr. Heubner has been gradually converted to my ways (*Berlin. Klin. Woch.*, 1895, No. 10, and frequently afterwards).

Indeed, so far has the balance swung in my favor that adepts of what they consider a new era, like Gregor (*Jahrb. f. Kinderh.*, Vol. XXX.) have gone too far in the direction of the active valuation of amylaceous foods in the nutrition of infants. But this excess of zeal will correct itself. For the present that excess has its many sources. It is caused by the failure of studying, or the neglect of, our own history by the over-specialization of medical practice and research which creates undue division of labor and artisans but no physicians; by the rivalry of "schools" that means nowadays the clinical laboratories of different universities bent upon swelling the outputs of the

printing presses—by the ambition of clinical teachers which tempts immature students to embark in a special little "study" in a workshop which for the time being and sometimes, for a long time to come they take to be the world, while it is their den only.

No wonder that literature is full of unripe products of priority claims, of loud cackling over the newly laid eggs. What does that hen of Claudius say:

"Erst leg 'ich meine Eier,
Dann recensir 'ich sie."

Now, then, far from feeling "cool," I feel warm at the turn things have begun to take. Infant diet will sometime, if not now, be a branch of medicine or rather hygiene. No longer controlled by the hundred different dicta of a hundred different dictators or interested manufacturers and stock owners, but governed by common sense and directed by a few well-understood facts, guided by the observation of the individual infant, with a due knowledge of the latitude kindly afforded by nature, and a proper regard for the physiology of the infant's alimentary tract.

One of the latest contributors to the milk-sugar literature is Joseph Prechtl.* His objection to selecting milk-sugar as an addition to the infant food is possibly based on the non-identity of the casein of cow's milk and that of woman's milk, on account of which milk-sugar must necessarily act differently on the two substances. Now casein is kept in solution by phosphate of calcium, the phosphoric acid of which is separated from the calcium by the action of lactic acid. When this is in excess and decomposes the active phosphate, casein is thrown out of its solution and loses its ready solubility.

Cow's milk contains three times as much casein as does human milk and much less milk-sugar (3 to 5, 6). In this relative proportion cow's casein remains in solution. When this proportion is disturbed by adding an undue quantity of milk-sugar to cow's milk, its casein becomes more indigestible through coagulation. It is disturbed by adding more than the quantity of sugar found in human milk, which contains so much more of it than cow's milk. It should have struck the sticklers for the action of chemical scales in preference to that of infant

**Jahrb. f. Kind.*, 53, 1901. "Is milk-sugar a proper ('vortheilhafter') admixture to infant milk?"

bowels, that the proper quantity of sugar to go with a cow's milk mixture would be the relative quantity met in cow's milk which keeps cow's casein in solution, and not the percentage of milk-sugar as contained in woman's milk which is in excess in its relation to cow's milk casein. It is only woman's casein, that though in a percentage three times smaller than that which is contained in cow's milk is not thrown out by its larger quantity of (milk-sugar born) lactic acid.

Artificial food requires the addition of sugar. It is customary to add to cow's milk milk-sugar enough to obtain the normal percentage contained in woman's milk. Why this is doubtless a wrong procedure I have just shown. Plain water dilution, which I abhor, would require 6 per cent.; cereal decoction admixtures demand less (certainly 1 per cent. less) because of their percentage in carbohydrates. This observation should commend itself to those in whose opinion the mutual exchange of different carbohydrates is a matter of course from a chemical point of view.

Milk-sugar, being a constituent of human milk, is recommended for admixture. Some of the proprietary foods, however, contain grape sugar because the manufacturers remind us that every sugar is changed into grape sugar. Cane sugar is most accessible and serves the same purpose, is of the same composition as milk-sugar ($C_{12}H_{22}O_{11} + H_{22}O$), and is, like the rest, finally changed into lactic acid. Soxhlet, who is a chemist only, though a great one, and Heubner and Hofmann exceed the 6 per cent. considerably, intending to replace the insufficient fat by another carbohydrate,* to great advantage to the doctrinarism of the calory theory, but to the detriment of the bowels.†

Epstein, however, found that no kilo baby should have more than 12 grms. (3 drs.) of milk-sugar, always with the

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* There is too much ado over the difference of the fat percentage in the two milks. It is 3.7 per cent. in cow's milk, 3.8 per cent. in woman's milk, according to reliable analyses.

† P. Zweifel ("Etiology, Prophylaxis and Therapy of Rachitis," 1900) believes he has found that the mixture of equal parts of milk and a 6 per cent. solution of milk-sugar is slightly more digestible than pure milk. In this respect, however, milk-sugar is inferior to chlorid of sodium. For the same author found that a 0.2 to 0.6 solution of salt added to an equal quantity of milk increases its digestibility to a still greater extent (p. 127).

proviso that the baby is in good health. Dyspeptic infants tolerate very much less.

In his preference of milk-sugar to any other kind of sugar Soxhlet is very eloquent. He is a chemist, and in that capacity is bound to know that most of it arrives unchanged in the small intestine where most of it is changed into lactic acid, but is not necessarily aware of the fact that it may cause diarrhea (indeed, many practitioners employ milk-sugar for its purgative effects), tympanites and colic. A chemist only, no physiologist, will calculate and assert the equivalence of carbohydrates, *i.e.*, sugar and fat, in the nutrition of infants. Where there is a purgative action there can be no nutritive effect. Besides, babies are no chemists, and not bound to be guided by chemical formulæ in their compound physiological functions.

Large quantities of milk-sugar cause diarrhea, as I, Neumeister and many others have observed. Experience taught me what I have always, more than forty years, recommended; that is, a small amount of sugar, even cane sugar, dissolved in warm water is one of the mild but effective purgatives when indicated for constipated babies.

For its purgative effect sugar is given to the newly-born in warm water or in some warm, aromatic tea. Such a medication is rarely demanded, for meconium is not often so solid or the mucus of the colon so inspissated as to require dilution. Still, there are cases with indications for a mild purgative. But A. Keller, (the accomplished assistant at Czerny's clinic, Breslau,) declares sugar to be by no means uninjurious or indifferent. That is why both these gentlemen oppose the employment of sugar in treating or feeding the newly-born, and advise—saccharin instead, "to which there can be no serious objection."* They do not deem it permissible to give anything but "tea" with saccharin the first day of life, because experiments with feeding at that time may give rise, sometimes, if not always, to "intestinal diseases." Why the latter assertion? Forsooth, because "Jansen ('Ergebnisse dev allg. Pathol. v. Lubarsch u. Ostertag.,' 1897, p. 826) made experiments on newly-born

* One objection may be the excessive sweetness of saccharin. It should not be forgotten that milk-sugar is less soluble and much less sweet than cane sugar; moreover, that colostrum is less sweet than milk.

calves with boiled milk which almost always resulted in hemorrhagic diarrhea." *

Formerly (formerly only?) the question of nourishing the newly-born was treated from a different point of view. It was deemed cruel and injurious to permit the usual loss of weight; means were recommended to improve the secretion of mother's milk or substitutes were anxiously searched for. Now, however, we are told that all the observations of centuries and all the thinking of obstetricians and practitioners were for naught, because a single man's experiment on calves, instituted in 1898, resulted in hemorrhagic diarrhea—in calves. These calves have changed it all. The human new-born babies when losing weight were considered to be the victims of unfavorable circumstances, of absent or deficient milk, of ill health of the mother, etc. Now they are just where they ought to be; when a week old some outside experimenter on calves considers your baby should weigh ten or fifteen ounces less than at birth. *Risum teneatis amici.*

Woman's milk contains after the first week of lactation, more milk-sugar than cow's milk. In the first week the percentage is low. Söldner† found 2.22, 3.13 per cent. until the sixty-first hour of life. G. Edlefsen,‡ 59 per cent. on the third day. Pfeiffer,‖ 3.36 at the same time. On the eighth day the percentage rises to 6, after that time very little. According to Söldner it amounts till the 170th day to 6.39, never in his experience beyond 6.87. As a matter of fact, however, it should be remembered that the milk of different women at various periods of lactation, and under varying circumstances is subject to changes. Nature is not given to fanatical doctrinarism like our omniscient chemists who regulate diet and training from behind the counters of their laboratories.

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* Czerny and A. Keller, "Des Kinder Ernährung, etc., 1901, p. 7."

† Z. f. Biol., 1896, Vol. xxxiii.

‡ Münch. Med. Woch., January 1, 1901.

	Tot. Alb.	Casein.	Alb.	Fat.	Sugar.
3 days	2.695	1.810	0.885	3.225	3.590
12 "	1.875	1.160	0.715	3.035	5.150
48 "	1.000	0.440	0.560	3.640	7.060
103 "	0.843	0.375	0.468	3.415	5.835
116 "	0.835	0.310	0.525	4.105	5.950

‖ Jahrb. f. Kind., xx., 1883.

It would do our Soxhlets and smaller men also, a world of good if they would see now and then an actual living baby. Said Heubner, a short year ago,* "*We have luckily advanced so far as no longer to believe in our dependence on laboratory experiments in the feeding of infants. For decades we have suffered from the fact that we were too eager to apply the experiments of laboratories in practice.*"

It is worth while to repeat that the colostrum of the first days while containing a great deal of albuminates, holds but little sugar, even less than is found in cow's milk. That is why the excess of sugar, to which the newly-born has to submit when water is given or artificial food, is not in accordance with what the newly-born has a right to expect.

If it be given milk-sugar, however, it should be taken into account that, as has been said before, the milk-sugar of human milk is probably not identical with that of the cow (A. Schlossmann, "Unterschiede Zwischenkuh und Frauenmilch," Leipzig, 1898), and that the milk-sugar of the market is very often impure.

In connection with that question, E. F. Brush ("Milk," 1898, p. 63) says: "In regard to the using of commercial sugar of milk as an addition to cow's milk for infant feeding, I think it is a mistake as there are undoubtedly all the other crystallizable milk-salts with the milk-sugar, and consequently we can know very imperfectly what we are feeding an infant with when we are giving milk-sugar. If the milk from which the sugar was crystallized contains improper vegetable salts, these would undoubtedly become crystallized with the sugar, and many of the proper salts would have become changed to the lactates, therefore, I think if sugar is to be used at all, although I deem it of doubtful necessity, the pure cane sugar is undoubtedly the best, because you know just what it is."

Amongst the clinicians who prefer cane sugar to milk-sugar and see "no inconvenience in so doing," is Marfan. His first reason for this choice is the frequency of adulteration of the milk-sugar in the market. In addition to the intestinal and nutritive disorders caused by an excess of lactic acid resulting

* Verein f. Inn. Med. Session of July 2, 1900. And many years before him Flügge plaintively expressed his regrets that "we have allowed ourselves to be guided by people who are neither hygienists nor physicians, but chemists, farmers or apothecaries."

from inordinate milk-sugar feeding, there are other dangers. Excess of lactic acid appears to have detrimental effects on the nutrition to such an extent that rickets has been explained by its chemical action. Like acetic, oxalic, formic acids, lactic acid has been claimed as the cause of rachitis by Ch. Heitzmann, in 1873; Tripier, in 1875; Neiss, in 1876; Siedamgrotzky and Hofmeister, in 1879, while Albarel could not verify their observations. Such differences had been noticed before. Schmidt and O. Weber had long ago met lactic acid in the bones of animals fed on that material; Marchand and Gorup-Besanez, in the urine; while Virchow and Lehmann found the bones and the blood alkaline. After all, however, it should be remembered that rachitis means more than merely excessive elimination of lime by the kidneys and intestines; its pathology is not complete without the soft swelling of the peri-epiphyseal cartilage, of the epiphyses and of the periosteum, also deformities of the bones. That is why the presence of lactic acid in the circulation should not be accused of being the cause of rachitis. But this much is certain, that by an undue presence of lactic acid the amount of phosphate of lime in the urine and in the feces, at least in one of them, is at once vastly increased, and that the bones are deprived of part of their calcium.*

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* It is on account of this and its eliminative, chemical action on lime (and thereby diuretic effect), which it removes in the shape of salts, that Rumpf gives lactic acid in those cases of angina pectoris which depend on calcification of the blood-vessels, about 15.0 grms. daily for months in succession. It is self-understood that the food should be fairly free of lime. That is why the diet is as follows: 250.0 meat; 100.0 bread; 100.0 fish; 100.0 potato; 100.0 apples; (or instead: green beans, peas, or cucumbers.)

INFANTILE SCURVY AND MARASMUS.

BY FRANCIS HUBER, M.D.,

New York.

In presenting an isolated case of infantile scurvy to the notice of this Society, which, through the Collective Investigation Committee, has done so much to add to our knowledge of the condition, an explanation and apology are necessary. The two photographs (Figs. I. and II. herewith shown) will serve both purposes.

The history briefly is as follows (that of the family not being reliable or important is omitted).

Anton Z., thirteen months old, was admitted to the "Jacobi Ward" Roosevelt Hospital, August 8, 1900. The patient, born in New York, is the seventh child and was as far as we could make out, of normal or average weight and development until about three months old. Then he is reported to have lost flesh and strength and as he did not improve, he was taken from the breast. It was claimed that the mother's milk was poor in quality, deficient, etc. At the age of four months he was fed on condensed milk; this plan was continued for two and one-half months. During the past two months he was given cream and water. The exact proportions or quantity could not be determined. The only other material fact elicited was that in spite of more or less gastroenteritis, nourishment was always taken eagerly. No other positive or reliable details could be obtained, though the mother was closely and carefully cross examined. The present illness is supposed to date back about three weeks before admission. About this time a number of hemorrhagic spots were observed upon the abdominal wall at the lower part. The gums began to bleed about a week before he was admitted to the hospital and at the same time spongy swellings were observed.

The appearance of the baby on admission was deplorable. The photograph presented, (Fig. I.) shows more plainly than

any attempt at description, the evidences of criminal neglect and the lesions of the extensive integumental ecchymoses. Emaciation was extreme and prostration marked. When the patient was stripped, the sunken eyes, the retracted abdomen, the partly closed lids, the immobility, the extensive black and blue discoloration of the body, and the marked emaciation in general, reminded the observer of the corpse of an infant, who had died of marasmus, undergoing the process of decomposition. The realistic impression was increased by the cadaveric odor.

Temperature was 95.4° F.; pulse, 93, weak and hardly perceptible; respirations, 20, shallow and weak. Cry feeble; gums swollen, spongy and bleeding readily; tongue and buccal

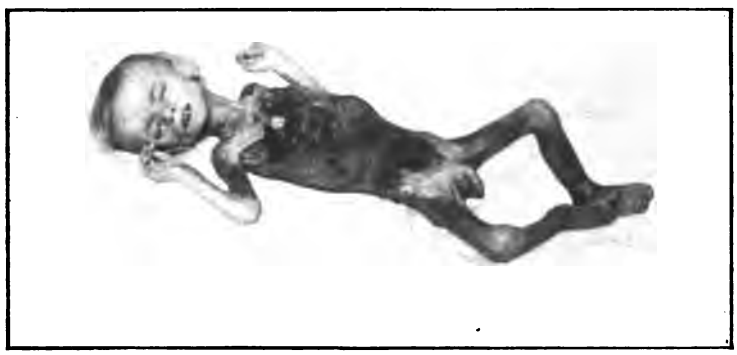


FIGURE I.—CONDITION OF THE BABY ON ADMISSION TO THE HOSPITAL.

mucous membrane raw and congested; belly retracted; fontanelle depressed. High degree of anemia with sallow complexion. Moderate degree of rickets present.

The anterior aspect of chest and abdomen was covered with large hemorrhagic infiltrations, principally integumental, although a few implicated the subcutaneous tissues. Smaller hemorrhages were found scattered over the rest of the body. There were bed sores over the spinous processes and the sacrum. There were hemorrhages into and ulceration of the lobe of the ear. The right knee was somewhat swollen, with the swelling above the joint involving lower third of femur. The general appearance is excellently portrayed in a photograph (see Fig. II.) taken by one of the house staff, a few days after admission when the

little one's condition had somewhat improved. Urine was pale, turbid, and neutral, with a specific gravity of 1,003; a trace of albumin; no sugar was found; triple phosphates were present.

Physical examination, negative; weight 8 pounds 1 ounce.

Blood examination August 15th. Considerable variation in size and color of red cells; white not materially affected. Red cells, 3,740,000; white, 6,700; hemoglobin 46 per cent.

TREATMENT.—Orange juice, whiskey in small amounts; water internally and salt solution per rectum to fill the blood-vessels and supply the necessary fluids. Milk (boiled) and bar-

FIGURE II.—SHOWING THE IMPROVEMENT IN THE BABY'S CONDITION.

ley water (1-4) in small quantity hourly, gradually increased. Later mutton broth with rice or barley water, soft boiled egg and scraped meat or a piece of steak to suck.

After the first few days, though the child was improving in every way, raw milk was tried. It was refused by the patient, though every other food was ravenously and eagerly taken. Furthermore, in view of the very hot weather, it was not considered wise to force the use of raw milk.

The precarious condition necessitated great care in feeding in the beginning. Milk with barley water was given in one drachm doses at first, later on two tablespoonfuls were given every hour. The orange juice was continued. Improvement

was rapid and in a little over two weeks the increase in weight was nearly five pounds. The appetite was excellent and as the child cried a good deal and did not appear to be satisfied, eggs, broths of various kinds, scraped meat and steak to suck were allowed. Diarrhea of varying amount, occurred off and on and caused some loss of weight, which, however, was readily regained, when the bowels were under control. More or less elevation of temperature characterized these attacks.

In spite of the extremely hot weather and the diarrhea referred to, the appetite remained excellent, in fact the child was always hungry, and took the food with the greatest relish. To omit details, it may be stated that the bad symptoms disappeared, the general condition improved, the hemorrhages gradually became less and all traces of the infiltration vanished. About five or six weeks after admission, the child was practically well, weighing 13 pounds 1 ounce.

A few general remarks regarding the care and the treatment may be in order:

Ordinarily when a case is presented with depressed fontanelle, weak pulse and general loss of fluids in the tissues of the body, the tendency is to supply the fluid by means of subcutaneous injections of sterile salt solution. Vargas (*Jacobi Festschrift*) has written a short article and presented photographs of patients suffering from marasmus, who were greatly improved by the subcutaneous use of artificial serum. The plan has been extensively employed in this city for several years. In the case before us, though the indication to supply water to the system was self-evident, the plan referred to was not feasible and was contra-indicated by the fragile condition of the blood-vessels and the numerous hemorrhagic infiltrations. No difficulty, however, was experienced in gradually introducing a sufficient amount of fluids by mouth and per rectum.

The benefit of fresh orange juice was quickly shown in our patient, a point noted by numerous other observers in their own cases. As a question of fact in mild cases, the therapeutic test of giving fresh fruits frequently clears up a suspected case.

The use of raw milk so generally advised and usually so very efficient could not be carried out, for though the child would take everything else in a ravenous manner, if raw milk were added to the rice or barley water the little patient would

persistently refuse the bottle and continue to scream until something else was substituted.

Though the case for the first few days did not appear to promise much, the use of orange juice, boiled milk and barley water, and later of scraped meat and broth, quickly conquered the vicious disturbance of metabolism and, in spite of the hot weather and a gastroenteritis, a cure was established.

209 EAST SEVENTEENTH STREET.

TREATMENT OF TUBERCULOSIS IN INFANCY AND CHILDHOOD WITH SPECIAL REFERENCE TO THE USE OF GUAIACOL.

BY B. K. RACHFORD, M.D.,

Cincinnati, O.

Tuberculosis in infancy and childhood is essentially a disease of lymphatic structures.

The tubercle bacillus finds its entrance into the body, as a rule, through the lungs and intestinal canal and is arrested by the tracheo-bronchial and mesenteric lymph nodes. This may be accomplished without injury to pulmonary or intestinal tissue. These nodes may hold the bacilli for an indefinite length of time, and as the number of bacilli increases, the contest for supremacy between the bacilli and the leucocytes goes on. In the great majority of instances these lymph nodes are a sufficient safeguard against the disease, and the bacilli are either destroyed or held captive, so that they can cause no material injury to the organism as a whole. But when the number of bacilli is overpowering, or when the contagion occurs in those who have inherited from tuberculous ancestors a type of lymph node tissue less capable of resisting the tubercle bacillus, the story is a different one. In these susceptible children the tubercle bacilli, finding entrance into these lymph nodes, are capable of producing great destruction of tissue, and as the unequal fight goes on, the bacilli escape into neighboring nodes, and chain after chain of mesenteric and tracheo-bronchial lymph nodes become infected, and an active and destructive tuberculosis is under headway. If the disease is not arrested other lymphatic chains become involved, the spleen is enlarged and a more or less diffused tuberculosis is established.

It is important for us, as physicians, to know that lymph node tuberculosis in childhood may be, and usually is, for a long time, a local disease confined to the lymphatics of some special part of the body. The tracheo-bronchial and the mesen-

teric lymph nodes are, however, the most important, not only because they are the most common sites of tuberculosis in infancy and childhood, but also because the disease may remain in these nodes for a long time, producing widespread destruction, without any very marked involvement of superficial lymphatics or without any of the ordinary symptoms of tuberculosis; that is to say, tuberculosis in infancy and childhood may be and usually is, for a long time, so concealed as to escape detection, if one depends for diagnosis upon the ordinary and well-defined symptoms of tuberculosis in the adult. This localized form of tuberculosis which may exist for months or years, gradually progressing to other and incurable forms, is the type of tuberculosis which is amenable to treatment, if it is recognized early enough and appropriate measures are adopted. But if the disease is allowed to progress and become more general, there is the ever present danger that intestinal tuberculosis, meningitis, or acute miliary tuberculosis will develop and place the patient beyond all hope of recovery.

The prognosis, however, of tuberculosis in childhood is, on the whole, very much better than tuberculosis in the adult. Very many of the cases of tuberculosis that one meets in private practice, among children, are amenable to treatment. This is true, not only of the cases of localized lymph node tuberculosis, but also of tubercular peritonitis and the milder forms of intestinal, pulmonary and chronic diffused tuberculosis, and most important of all is it, that these cases can as a rule be satisfactorily treated *at home* without the aid of the climatic and other advantages afforded by favorably located sanitariums.

The above statements emphasize the importance of the early diagnosis of concealed tuberculosis, and for this reason a short *résumé* of the symptomatology of this condition is here outlined.

In the *New York Medical Journal*, August 10, 1895, in a paper on "The Diagnosis of Concealed Tuberculosis," I dwelt at length upon the symptomatology of this condition, and many of the points in the following outline are taken from that paper.

SYMPTOMATOLOGY OF TUBERCULOSIS OF THE LYMPH NODES.—Experience leads the physician to suspect this condition when he is confronted with a certain type of child. It is difficult to draw a mental picture of the appearance which the tuberculous child presents to the trained eye of the physician. These children, as

we see them in private practice are, as a rule, nervous, irritable, undersized, slender-limbed, oval-faced, fair-haired and dainty little creatures, and we are at once impressed with their frailty. They have bright eyes and thin, transparent, fair skin, which makes them very attractive. There is another and less common type of the tuberculous child which is usually described as having a heavy figure, thick lips and hands, opaque skin and large thick bones. We do not depend, however, upon these types to make a diagnosis, since there are many important symptoms which point more or less clearly to concealed tuberculosis.

Anemia without apparent cause is very suggestive of tuberculosis of the lymph nodes. The progressive anemia which marks the progress of this form of tuberculosis is a very pronounced symptom, and is much more characteristic of the disease as it occurs in the infant and child than it is in the adult.

The anemia grows apace as the disease spreads through the lymphatic tissues, until finally it becomes very extreme. If one can eliminate malaria, syphilis, rheumatism and intestinal disorder as causes for the anemia, one is justified then in suspecting a concealed tuberculosis, even though there be no other symptoms to assist in the diagnosis. When, in an instance of this kind, one can find a family history of tuberculosis, and also, which is of equal importance, a history of exposure to the tuberculous contagion, one is then quite justified in making a provisional diagnosis of tuberculosis and at once instituting proper treatment. If in addition, however, one learns that the child has lost in weight, or has even failed to gain in weight, which in the young child has the same significance that loss of weight in the adult has, then we have established another link in the chain of circumstantial evidence. Further inquiry may perhaps develop the fact that the child is excessively nervous and that it is suffering from night terrors or nocturnal incontinence of urine.

Precocious anemic children suffering from general nervous irritability should always be searched for other signs of concealed tuberculosis. By still further inquiry one may learn that the child suffers frequently from mild attacks of bronchitis, that it catches cold readily, and may also have pain in the side on slight exercise. This last symptom may be accompanied by dyspnea and rapid heart action. The pain in the side on exer-

cise, however, and the proneness to catch cold are oftentimes most significant and important symptoms in the early diagnosis of tuberculosis in childhood.

Dyspepsia and diarrhea, obstinate in character, and unaccounted for by errors in diet, are also common symptoms of this form of tuberculosis. This is especially true of the disease as it occurs in the infant. A history of feeble digestion, frequent and obstinate attacks of diarrhea, sometimes alternating with constipation and associated with enlargement of the spleen, and possibly of the liver, presents a symptom group which at once suggests mesenteric tuberculosis. These are the cases which afterward develop enormous and board-like abdomens, with contracted chests, flaring ribs and emaciated bodies, which give us the well-known picture of tabes mesenterica.

It is well for us, however, to remember that many of these cases of tuberculosis in the infant and child may have a long-continued and wasting diarrhea without actual ulceration of the intestine, and are therefore amenable to treatment. But again before leaving this symptom group I wish to emphasize the fact that enlargement of the spleen is a common accompaniment and a most important sign of acute tuberculosis in the infant and child.

It is important to note that all of the above symptoms may exist without any very pronounced change in the temperature of the body. It may be, however, that after our attention has been called to the possible existence of concealed tuberculosis that we will find, if the temperature be carefully recorded over a number of days, that occasionally there will be subnormal temperature in the morning, or a slight rise of one or two degrees in the afternoon.

These tuberculous children, however, may have over a considerable period of time, an afternoon temperature reaching from 102° to 104°, and yet be upon their feet and protest that there is nothing the matter with them. Fever of this character without the ordinary accompanying discomforts, is suggestive of tuberculosis. The temperature, however, of patients suffering from tuberculosis of the lymph nodes is by no means an indication of the progress and extent of the disease. The presence of external lymph node enlargement is of itself not indicative of deep-seated disease. The gradual increase in size, however, of superficial lymph nodes, especially in the neck and groin, may,

when associated with other symptoms above recorded, be significant.

In older children the appearance of any of the preceding symptoms with failure in health and asymmetry of development are suggestive of tuberculosis.

In hospital practice I have for a number of years been in the habit of using tuberculin for the purpose of confirming a diagnosis of lymphatic tuberculosis, beginning with ¼ to ½ mgs. and increasing to 2 mgs., if no reaction was obtained from smaller doses. I have witnessed no bad results from this practice, yet I have not thought it expedient or necessary to make use of it in my private practice.

It is also well to rememb r that the diagnosis of obscure tuberculosis may sometimes be confirmed by the appearance of tub rcles in the retina.

It is not my purpose to discuss in a general way the symptomatology of tuberculosis. I have endeavored in the above outline simply to call attention to a group of symptoms no one of which is characteristic of tuberculosis, but which taken collectively as they appear in various groupings in cases of early tuberculosis of the lymph nodes, are sufficient to aid us in arriving at a diagnosis.

I have not dwelt upon the interesting group of pressure symptoms which result from the presence of enlarged lymph nodes impinging upon nerves, arteries, veins, bronchial tubes and other structures in various parts of the body which often assist us in locating the diseased structures.

Neither can I touch upon the importance of a large number of other and more advanced tubercular manifestations, such as caseation of lymph nodes, cold abscesses, eczema, phlyctenular ophthalmia, corneal ulcers, suppurative inflammation of the eyelids, otorrhea, mastoid disease and inflammation of bones, joints, lungs and other structures.

TREATMENT.—The keynote to the treatment of tuberculosis in infancy and childhood is to maintain nutrition by a proper diet. This may be said to be the most important indication in the treatment of this disease at all ages. But the importance of nutrition becomes more and more paramount the younger the patient, and in artificially fed infants the problem presented is one of the greatest difficulty.

The tuberculous infant under one year of age that does not

happen to have a non-tuberculous mother from which to draw its breast-milk is indeed in a bad way. These children, as has been previously noted, have feeble digestions and suffer frequently from diarrhea. They cannot, as a rule, digest the casein of cow's milk, and, therefore, cannot be properly nourished with artificial foods. A wet-nurse is, for these reasons, almost absolutely necessary for the proper management of young tuberculous infants; and even after the first year of life I have found it necessary, as a rule, not to depend exclusively upon cow's milk, otherwise casein indigestion and diarrhea will bring disaster.

We may, however, when a suitable wet-nurse cannot be obtained, be forced to artificial feeding. Under these conditions I have sometimes used with benefit certain proprietary foods, after failing to keep up the nutrition of the infant with modified cow's milk. Nestlé's food and malted milk or a combination of the two have often served me a good purpose in the management of these cases.

I ordinarily begin by giving Nestlé's food alone. When the stools become normal under this treatment, cod-liver oil, in the form of an emulsion, or the clear cod-liver oil, is added to several of the feedings, until an infant, a year or a year and a half of age, is taking from one and a half to two teaspoonfuls of cod-liver oil in twenty-four hours. After a time I also add the white of one egg to one of the bottles of Nestlé's food and the yellow of an egg to another. In this way after a few weeks of treatment the little patient may be in a condition to commence the gradual substitution of dilute cow's milk for the Nestlé food mixtures; but even after this substitution has taken place the cod-liver oil and the raw egg are still added to the milk mixtures. This is necessary, because these children for many months are not able to take a mixture stronger than one-half milk. It requires constant watchfulness to properly feed and nourish these infants.

In older children milk and cod-liver oil remain the foundation-stones of the treatment. Where good, pure, fresh cow's milk can be obtained it is, as a rule, well borne, and in children over three years of age it is well to urge the child to take at regular intervals as much of this food as possible. One of the important advantages gained by sending tuberculous children to the country is that there they may get clean, rich, fresh cow's

milk in unlimited quantities. The other food of the tuberculous child should also be carefully selected. He should be given fresh eggs, good beef and poultry, and to older children cereals, fresh fruits and well-cooked vegetables may be allowed. But all made dishes, sweets, pastries, and in short all foods difficult of digestion should be excluded from his diet. The dietetic treatment of this condition ranks above all other measures. Every attention should be given to it, and it should be carried out not only month after month but year after year until all evidences of the disease have disappeared.

The next most important agents in the treatment of tuberculosis in childhood are fresh air and sunshine. These can, as a rule, be obtained in the suburbs of even our largest cities, and in most of our smaller cities they can be obtained without any change of home whatever.

A number of years ago when I took charge of the children's wards at the Cincinnati Hospital, I prevailed upon the trustees of that institution to convert one of the corridors leading to my wards into an open-air apartment. This corridor is now so arranged that the beds can be rolled from the wards into it. In this way I am able to keep my tuberculous patients out of doors without increasing the nursing force of the wards. Infants and small children suffering from this disease are kept out of doors, by this arrangement, for a variable length of time during all seasons of the year. In the very hot weather of summer the beds remain in this open air ward night and day, only being brought in in stormy weather. In colder weather, however, the beds are brought into the wards at night and remain outside a greater portion of the day. In winter these tuberculous patients are hooded and covered with warm bed clothing, and if necessary hot water bottles are placed in the bed, and they spend from an hour to two or three hours in the open air every day. The length of time of exposure, of course, must be determined for each individual case.

Older children suffering from this disease are allowed to play in this open ward for a good portion of the day. The fresh air treatment of tuberculosis is, I think, even more important in the infant and child than in the adult, and if faithfully carried out will give better results. In private practice fresh air and sunshine can of course be obtained in a much more satisfactory way than in hospital practice.

Many of these cases no doubt will do better if this fresh air treatment be carried out in high altitudes having a low barometric pressure; but for infants and very young children, the comforts and conveniences of a suburban home in any of our large cities cannot, so far as the treatment of tuberculosis is concerned, be offset by high altitudes with poor home accommodations.

Exercise is not an essential part of the fresh air treatment. In fact, it is contraindicated in all acute cases marked by high fever or other symptoms indicating rapid progress of the disease. Patients of this kind are to be placed out of doors in baby carriages, beds or chairs, and are thoroughly protected from the weather by suitable clothing. In the more chronic forms of the disease not marked by high fever and other acute symptoms, gentle exercise and lung inflation may be a part of the fresh air treatment.

MEDICAL TREATMENT.—The special purpose for which this paper was written was to call attention to the great value of guaiacol in the treatment of tuberculosis in infancy and childhood. Guaiacol, in my opinion, far outclasses all other drugs in the treatment of this condition.

In May 1894, * I called attention to the great value of inunctions of guaiacol and recommended the following prescription:

℞ Guaiacol,..... 3 i
Lanolin,..... 3 ii
Lard,..... 3 v

M. Sig. One level teaspoonful to be rubbed into the chest at bedtime each day.

This prescription I have used for the past eight years in almost every case of tuberculosis in infancy and childhood which I have had an opportunity to treat, and the experience which I have had with this prescription, in many hundreds of cases, has convinced me of its great value.

It is a well-known fact that guaiacol is one of the few drugs which, when it is applied to the skin, is rapidly absorbed by the lymph channels, and is in that way carried into the general circulation, producing the physiological action of the drug. Its great value in the treatment of the lymph node tuberculosis of infancy and childhood in all probability depends upon the fact

* *Ohio Medical Journal*, May 1894.

that by inunction it can readily be brought into contact with the diseased lymph nodes, and in that way acts as a lymphatic antiseptic.

Inunctions of guaiacol, notwithstanding their great value in the treatment of tuberculosis of infancy and childhood, are of comparatively little value in the treatment of this disease in the adult. The reasons for this are evident. In the first place the general lymphatic and glandular systems are more active in the child than they are in the adult, and in the second place adult tuberculosis is not, as a rule, tuberculosis of the lymph nodes.

In acute tubercular conditions marked by fever and other active symptoms, I ordinarily direct that a level teaspoonful of the above ointment be rubbed into the skin over the abdomen and chest night and morning. The rubbing should be done gently and firmly and should occupy ten or fifteen minutes. This treatment may be continued for from one to two weeks and then one inunction a day may be continued for an indefinite length of time. It is well, however, after the fever and other active symptoms have been controlled, either to discontinue the inunctions for a while or to give them two or three times a week as long as it may be deemed necessary. This treatment is of the greatest value in all forms of lymphatic tuberculosis and even when long continued can do no harm.

In tubercular peritonitis the good results which follow the use of this treatment commence at once and the patient, as a rule, slowly but steadily recovers. I have frequently seen the distended, tender and board-like abdomen, which this disease produces, lose its tenderness, distension and tumidity to a degree which marked the establishment of convalescence within a period of three weeks.

In these cases when the active symptoms are in abeyance I frequently substitute carbonate of guaiacol internally for the inunction treatment. Carbonate of guaiacol has the advantage of being easy of administration, and when mixed with a little milk-sugar can be given in powder, without complaint from these whimsical little patients. The value of carbonate of guaiacol in the treatment of all forms of tuberculosis in infancy and childhood is very great, but it is especially valuable in the treatment of intestinal and mesenteric tuberculosis. Guaiacol holds first rank as an intestinal and as a pulmonary antiseptic. And it is possible that a large part of its beneficial action may

depend upon its power to control and destroy the streptococci, which are constantly associated with the tubercle bacilli in the destruction of tissues.

Creosote, which has for many years held first place among drugs in the treatment of adult tuberculosis, is also of value in the treatment of tuberculosis in infancy and childhood. It cannot, however, be used very satisfactorily as an inunction, and its disagreeable taste is a very serious drawback to its successful use in tuberculous diseases of children. Since tuberculosis is a chronic disease, and since children suffering from it are as a rule whimsical and self-willed, it requires a great deal of tact to medicate these children. The medicines must be so palatable that these little patients will take them without a struggle, otherwise the indulgent mother will soon discontinue the treatment. For this reason creosote is available only in well selected cases and in older children, when it may be used in connection with guaiacol inunctions. Children old enough to take a capsule may be given the following prescription:

℞. Tinct. Gentian..... ʒ iss.
Creosote (wood)..... ʒ ss.

M. Sig. Drop 3 to 5 drops in a capsule and take every six hours, followed by a drink of milk.

Inhalations of creosote are also of considerable value in the treatment of older children suffering from pulmonary tuberculosis. For this purpose the following formula may be used:

℞. Tinct. opii camph..... ʒ ii.
Creosote (wood)..... ʒ ii.
Alcohol..... ʒ iv.

M. Sig. 5 to 15 drops in creosote inhaler; use for fifteen minutes or more, three times a day.

Older children do not object to creosote by inhalation or to the creosote in capsules, so that these prescriptions may, under favorable conditions, be continued for a number of weeks without becoming distasteful to the patient. To infants and younger children, however, below the age of eight, I rarely attempt to give creosote in any form; this is because I have much more faith in the efficacy of guaiacol and it is much easier of administration. In chronic forms of tuberculosis or even in acute tuberculosis, after the active symptoms have been controlled by rest, fresh air, diet and the guaiacol treatment as

above outlined, cod-liver oil is one of our most valued remedies and should be given as a routine practice to all such cases. Mention has previously been made to the fact that it can be successfully administered to the infant, mixed with the food in the nursing bottle.

Iodid of iron, as well as other preparations of iron, are of value in treating the anemia of tuberculosis, after the disease has been brought under control by the treatment previously outlined. In the presence of fever, however, and other active symptoms, the iron salts, including the iodid, probably do more harm than they do good. The iodid of iron, however, has long enjoyed a favorable reputation in the treatment of the more chronic forms of glandular tuberculosis. This reputation no doubt is in great part deserved, as many of these patients are greatly benefited by this drug. Care, however, must always be taken not to allow it to disturb the digestion or injure the appetite since it is a cardinal rule in the treatment of tuberculosis that anything that interferes with the taking or the digestion of food, will, by interfering with the nutrition of the patient, do more harm than it can possibly do good.

Arsenic is a remedy also of value in the treatment of the most chronic forms of glandular tuberculosis. Enlarged tubercular lymph nodes will oftentimes grow smaller under its use.

Malt containing diastase may also be of considerable value in well selected cases. Taken after meals it will oftentimes assist digestion, and in that way promote the appetite and bring about a better condition of nutrition.

While it is not the purpose of this paper to discuss the surgical treatment of tuberculosis, mention may here be made of the facts that the removal of tuberculous bones and lymph nodes is not only at times expedient but is often necessary and laparotomy for tubercular peritonitis has been followed by very satisfactory results.

A NOTE ON THE LITTLE FINGER OF THE MONGOLIAN IDIOT AND OF NORMAL CHILDREN.

BY J. PARK WEST, M.D.,
Bellaire, O.

Five years ago Dr. T. Telford Smith called attention* to a peculiarity in the shape of the hand which existed to a greater or less degree in nearly every case of Mongolian idiocy he had examined, and consisted of a marked outward bowing or curving of the little finger. The skiagraph accompanying his paper shows "that the second phalanx of the little finger is considerably shorter than normal, and there is much lateral displacement of the terminal phalanx."

Two years ago a girl, two and a half years old, who presented many of the physical but none of the mental characteristics of the Mongol type of idiocy, came under my care. Her little fingers were somewhat curved, but not so much as those of her cousin, a normal male one month her junior. Of four Mongolian imbeciles seen previously but one had as much curve in the little fingers as this normal boy. This suggested the examination of the little fingers of other normal children, and after seeing a small number straight little fingers appeared to be the exception and a varying degree of curvature was quite common.

To form an estimate of the various degrees of curvature and the percentage of the different forms, I have within the past two years examined the little fingers of 605 children under twelve years of age, 296 being males and 309 females. These were children (with the exception of about 30 seen at a school) I had occasion or could make occasion to see in any way, and a number were seen several times. They were the ordinary children of an industrial community made up chiefly of American-born, with a few Bohemians, Russians, Germans, Italians and French. None were excluded except a few feeble-minded, a

* *Pediatrics*, October, 1896.

few with rheumatism, marked rachitis or deformities, and, after 50 for each year had been obtained, no more for that period were examined. Before the examinations had proceeded far it became evident that four varieties of little fingers existed in normal children, and for convenience they were recorded as follows, viz.: (1) Straight; (2) slight curve; (3) distinct curve; (4) marked curve. Of the 605 children, 112, or 18.5 per cent., had straight little fingers; 175, or 28.9 per cent., a slight curve;

Fig. 1.—STRAIGHT LITTLE FINGER.

199, or 32.9 per cent., a distinct curve; and 119, or 19.6 per cent., a marked curve.

The curving or bowing in this last class (Fig. IV.), corresponds in degree to that shown in the photographs of the idiots of the Mongol type published by Dr. Smith, and this skiagraph (Fig. V.), shows even more distinctly the same anatomical condition causes the curving in this feeble-minded type and in the normal child with the marked bowing of the little

Fig. II.—SLIGHT CURVE IN LITTLE FINGER.
Same in Ring Finger.

Fig. III.—DISTINCT CURVE IN LITTLE FINGER.

fingers. A few children showing the marked curve to the naked eye do not show this anatomical condition distinctly to the fluoroscope; but some in the third class, those with a distinct but not so marked curve, (Fig. III.), also show the short middle and more or less displaced distal phalanx.

It was not unusual to find the children of a family showing quite different degrees of curving in the little fingers, and 51 of the 605, or about 8 per cent., showed a different amount of

Fig IV.—MARKED CURVE IN LITTLE FINGER.

bending in these two fingers without previous disease or injury to account for it. In a small number, probably 2 per cent., there was a distinct curve in the ring finger as shown in the second photograph (Fig. II.) Five sets of twins were seen and only one pair had fingers alike.

Close examination for stigmata of degeneration was seldom made, but my impression is they were not present oftener in the third and fourth classes than in the other two. From my

knowledge of quite a number and from inquiries about others I believe the mental capacity of the four classes is about the same. Four children under two years of age with the markedly curved little fingers have been watched from birth. The mental and physical development of all has been fully up to the average, and in three of them there has been an increase of the curving.

I have seen but nine Mongolian imbeciles within the past four years. I assisted at the birth of two of these, and the ages

Fig. V.—SKIAGRAPH OF HAND OF FIG. IV.

of the others varied from nine months to twelve years. Only one had such a bowing in the little fingers as this fourth photograph shows. Two others had a less marked curving that was apparently due to the anatomical peculiarity mentioned. Three had very slight curving and two had straight little fingers. One, nine months old, seen for the first time ten days ago, had the right little finger straight and the left somewhat curved, and it

is likely this will increase as the child grows older. Of the two seen at birth one had straight little fingers, the other a very slight bend at the distal phalangeal joint that had almost disappeared before his death, seven months later. A conclusion cannot be based on these few cases, but there is a striking similarity in them and in the normal children.

I am indebted to my colleague, Dr. J. S. McClellan, for the skiagraph and for the privilege of examining the hands of several children with the fluoroscope.

AGE	SEX	STRAIGHT	SLIGHT CURVE	DISTINCT CURVE	MARKED CURVE	NUMBER
Under 1 year	Male	9	3	8	5	25
	Female	11 — 20.	5 — 8.	11 — 19.	3 — 8.	30 — 55.
Between 1 and 2 years	M.	9	10	4	5	28
	F.	7 — 16.	6 — 16.	7 — 11.	2 — 7.	22 — 50.
2 and 3 "	M.	8	10	7	7	32
	F.	4 — 12.	6 — 16.	5 — 12.	3 — 10.	18 — 50.
3 and 4 "	M.	5	5	12	4	26
	F.	5 — 10.	7 — 12.	6 — 18.	6 — 10.	24 — 50.
4 and 5 "	M.	2	11	6	8	27
	F.	2 — 4.	11 — 22.	7 — 13.	3 — 11.	23 — 50.
5 and 6 "	M.	3	4	14	4	25
	F.	6 — 9.	2 — 6.	10 — 24.	7 — 11.	25 — 50.
6 and 7 "	M.	2	5	10	5	22
	F.	3 — 5.	9 — 14.	12 — 22.	4 — 9.	28 — 50.
7 and 8 "	M.	2	6	6	6	20
	F.	5 — 7.	10 — 16.	8 — 14.	7 — 13	30 — 50.
8 and 9 "	M.	1	9	7	4	21
	F.	5 — 6.	14 — 23.	7 — 14.	3 — 7.	29 — 50.
9 and 10 "	M.	5	7	8	6	26
	F.	2 — 7.	4 — 11.	12 — 20.	6 — 12.	24 — 50.
10 and 11 "	M.	3	7	10	5	25
	F.	2 — 5.	12 — 19.	8 — 18.	3 — 8.	25 — 50.
11 and 12 "	M.	5	4	7	3	19
	F.	6 — 11.	8 — 12.	7 — 14.	10 — 13.	31 — 50.
TOTALS M.		296	175.	199.	119.	605.
TOTALS F.		309	112. (18.5 %)	(28.9 %)	(32.9 %)	(19.6 %)

DISCUSSION.

DR. ROTCH.—It seems to me this corresponds somewhat with the investigations of the criminal ear.

DR. KOPLIK.—In demonstrating this Mongolian finger I have long been impressed by the fact that many people can show a small bend in their fingers. The Mongolian idiot shows the bend merely a little more marked than normal children, and I am very much pleased that Dr. West should have worked out this subject.

GREAT FLUCTUATIONS IN TEMPERATURE IN THE TERMINAL STAGE OF PULMONARY TUBERCULOSIS.

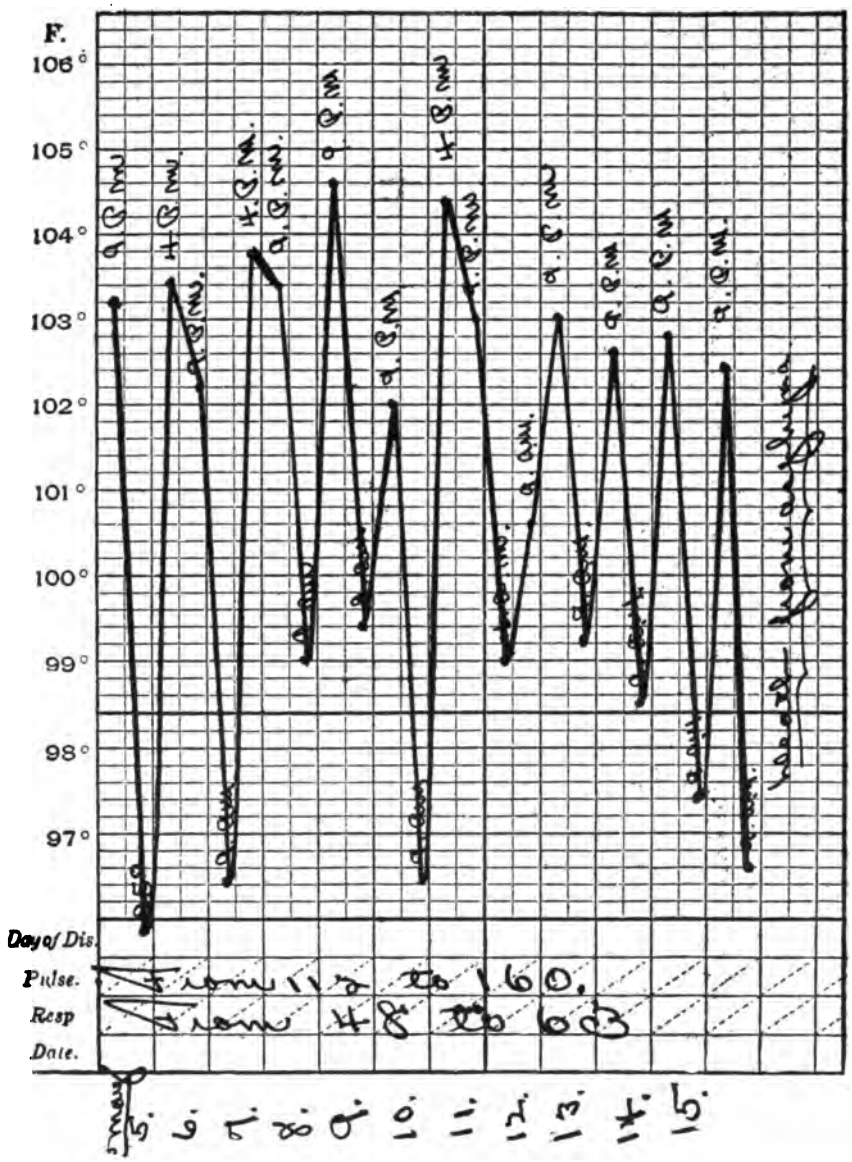
BY SAMUEL S. ADAMS, A.M., M.D.,

Washington, D. C.

The following cases of pulmonary tuberculosis, with the temperature charts, are presented to illustrate the apparent harmlessness of wide fluctuations of temperature in the terminal stage of the disease. While the rise and fall of temperature resembles in some respects the fever of sepsis, nevertheless it differs from it in not being accompanied by chill, sweat and great prostration. The symptoms of septicemia and pyemia are almost identical in the child and adult, and the high temperature is often associated with mental disturbances of more or less severity. In the terminal stage of pulmonary tuberculosis, however, the temperature may reach 108° F., and in a few hours drop to 95° F. without any apparent effect upon the child, either mentally or physically. Indeed, such wide variations have occurred in some of our cases without disturbing their appetite, disposition, intelligence or play. It is not unusual to see a child enjoying his breakfast with a rectal temperature too low to be registered by the thermometer; and later in the day find him sitting in bed eating his evening meal with relish, in spite of the fact that his rectal temperature is now 106° or 107°. The absence of evidence of collapse in the first instance and of the usual phenomena of hyperpyrexia in the second, can only be explained upon the assumption that some irritant acts upon the heat centres differently from that present in purely septic conditions.

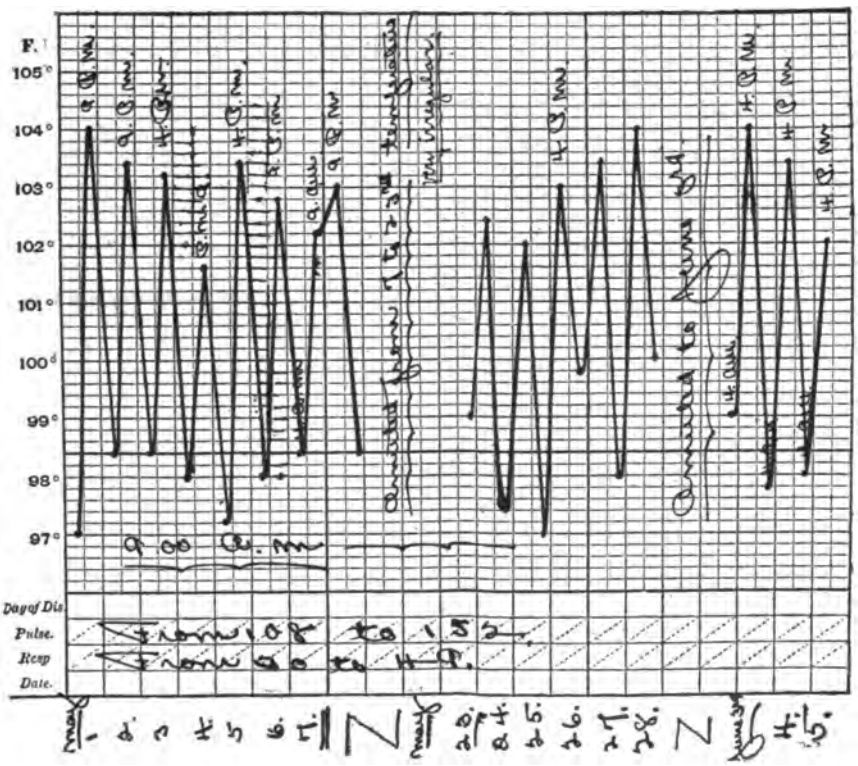
CASE I. GENERAL MILIARY TUBERCULOSIS.—James J., colored, aged five years, admitted to hospital April 8, 1891, and died May 15, 1891. Had measles three months before admission, which was soon followed by cough, rapid emaciation, and loss

Chart of Case I.



of strength. Both apices involved. The great fluctuations in temperature did not disturb him. He had neither chills nor sweats, and high temperature was not attended by any nervous manifestations. He seemed equally contented whether the bodily temperature was very high or very low. The necropsy confirmed the diagnosis of marked infiltration at the apices of the lungs, with general lymphatic and visceral involvement.

Chart of Case II.



CASE II. PULMONARY TUBERCULOSIS.—A. B., aged seven years, colored, female, was admitted to the hospital March 15, 1891, and died July 14, 1891. Her previous history was indefinite, it being stated that her present illness began two months prior to admission with severe cough, fever and rapid emaciation. She complained of pain over left chest during violent paroxysms of coughing, but was at other times composed. There

was never any symptom referable to the temperature. A cavity of moderate size was located in upper part of the left lung, when she was first examined, and, as the disease progressed, areas of consolidation throughout the lung.

The necropsy revealed a cavity two inches in diameter at the apex and several small ones in the left lung, which was also

Chart of Case III.

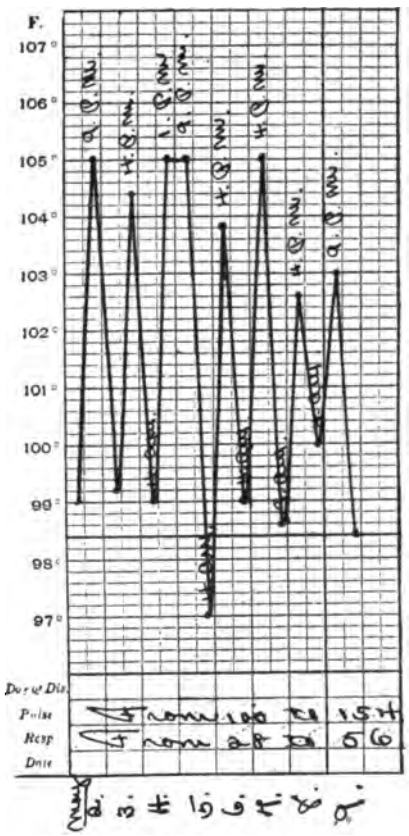
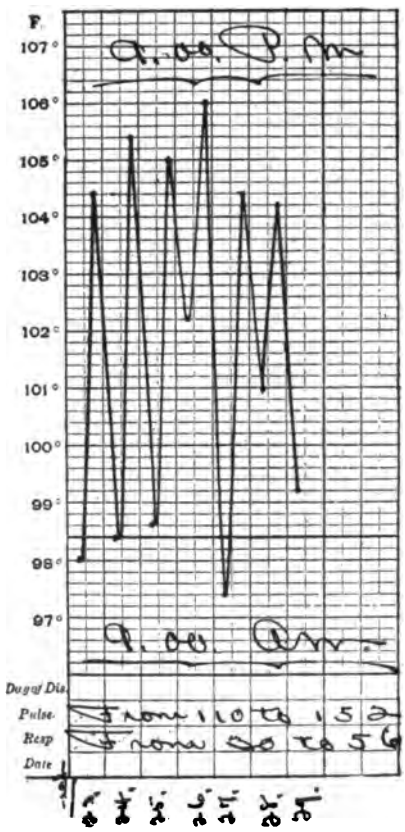


Chart of Case IV.

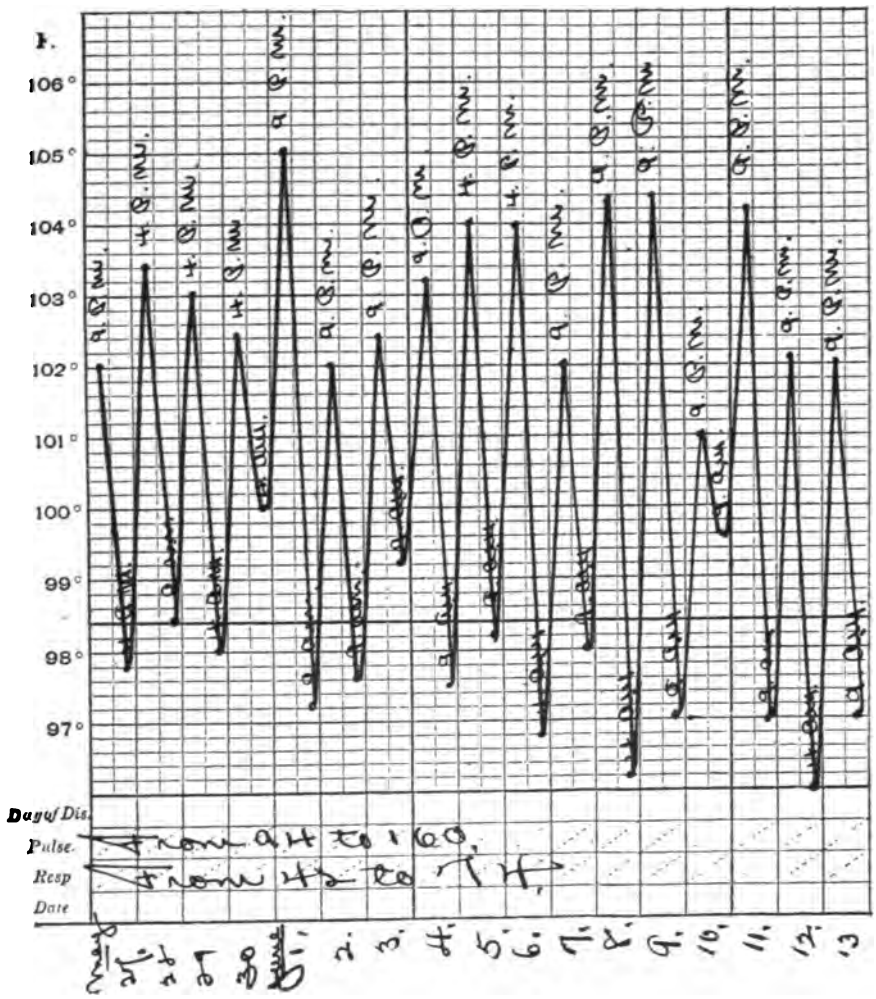


consolidated in many places. The right lung was congested and contained a few miliary tubercles.

CASE III. CARIES OF SPINE, WITH TUBERCULOSIS OF RIGHT LUNG.—Joseph B., colored, aged eleven years, was admitted to hospital May 2, 1891, and died May 10, 1891.

Father and mother died of "phthisis." Origin of disease one year ago. Since then had had cough and fever, and has emaciated. Had marked cervical deformity. Consolidation of entire right lung. Most of his suffering was from dyspnea. His mind was clear at all times, whether his temperature was 105° or 97°. No necropsy.

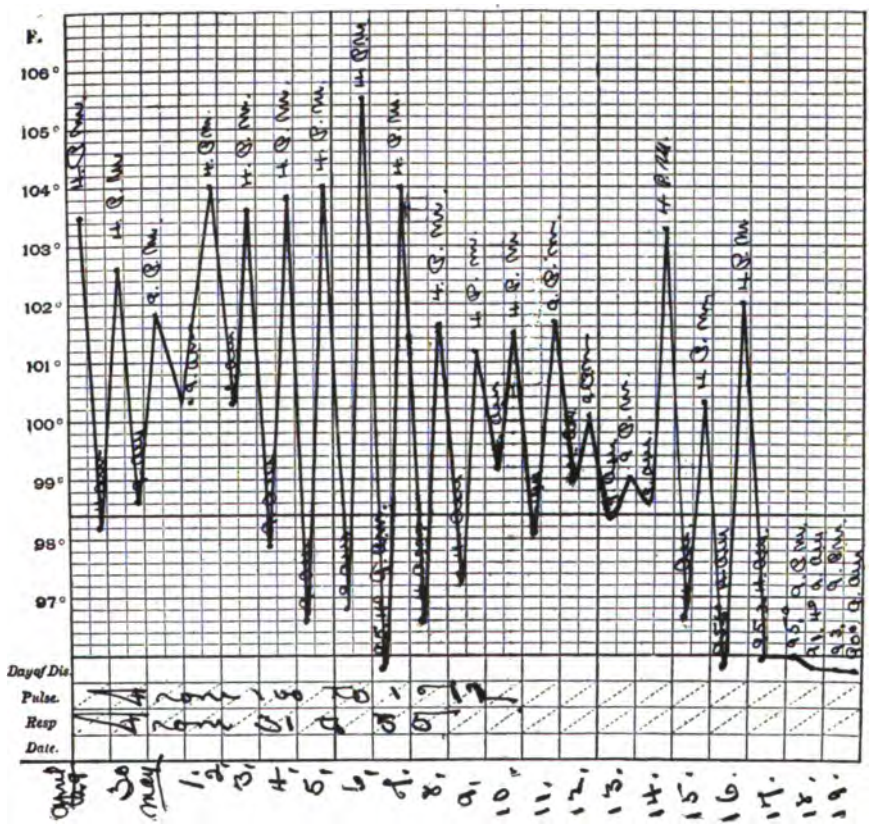
Chart of Case V.



CASE IV. PULMONARY TUBERCULOSIS.—Kate J., colored, aged three years, was admitted to the hospital March 23, 1897, and died a week later.

Paternal and maternal tubercular history positive. Onset of her disease so insidious that its beginning could not be ascertained, though of several months' duration. Tubercular process

Chart of Case VI.



general throughout both lungs. Suffered from severe cough, dyspnea, and pains in head and abdomen at times. Had neither chills nor sweats, nor did the fluctuations of temperature affect her sleep, appetite or intelligence.

Necropsy not permitted.

CASE V. PULMONARY TUBERCULOSIS.—Richard G., white,

aged eleven years, was admitted to hospital April 27, 1894, and died June 13, 1894.

Illness began seven months before with cough, fever and loss of flesh and strength. Right lung chiefly involved. All signs of a cavity, including "cracked pot" sound at right apex. All through illness he seemed comfortable, with the exception of pains in the abdomen, caused by enteritis. No delirium, chills or sweats. His intelligence was not affected by the variations of temperature.

Chart of Case VII.

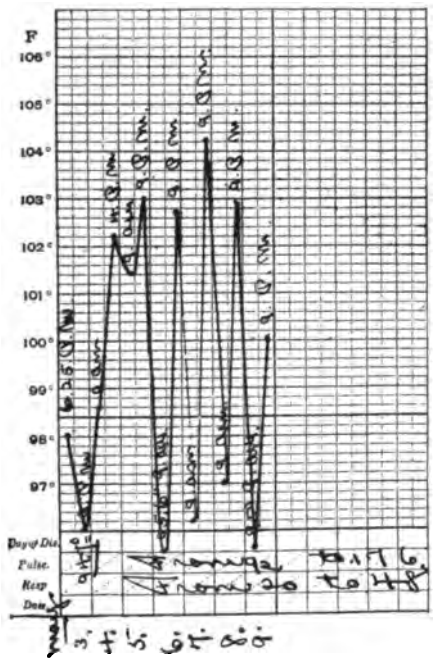
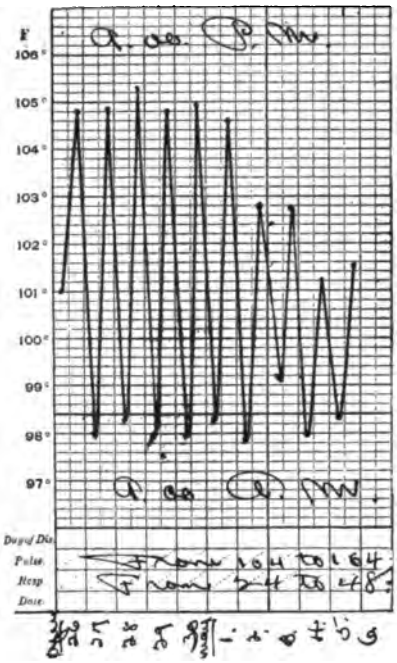


Chart of Case VIII.



The necropsy revealed destruction of almost the entire right lung; a sac, whose walls were composed of the thickened pleura, remaining. General miliary tubercles in other organs and lymph nodes.

CASE VI. GENERAL TUBERCULOSIS.—Edward C., colored, aged four years, was admitted to the hospital August 5, 1899, and died May 19, 1900. He was admitted to the ophthalmological service for "scrofulous conjunctivitis," but shortly after admission there were evidences of general miliary tuberculosis,

with prominent pulmonary lesions. A cavity was soon discovered at the right apex. During his long illness he exhibited no subjective symptoms, but was always quiet and contented. His appetite was always good. A fluctuation of nine or ten degrees in his temperature had no apparent effect, as there was neither chill, fever nor delirium.

The necropsy revealed general miliary tuberculosis, a good-sized cavity in the right apex, and areas of consolidation throughout both lungs.

CASE VII. PULMONARY TUBERCULOSIS.—Lina R., colored, aged eight years, was admitted to the hospital May 2, 1901, and died eight days later.

Present illness began three months previous with cough, fever and gradual emaciation. Both lungs involved, especially the left apex. Dyspnea, severe headache and pain in chest. No delirium or other nervous manifestations; neither chills nor sweats. Sputum contained tubercle bacilli. Marked leucocytosis, chiefly polymorphonuclears.

The necropsy showed general miliary tuberculosis. Both apices consolidated, but no cavities. Bronchial lymph nodes much enlarged.

CASE VIII. PULMONARY TUBERCULOSIS.—Rachael G., colored, aged eight years, was admitted to the hospital January 11, 1901, and died May 6th following.

This child was admitted for a burn of the second degree, but was soon transferred to the medical service. A cavity as large as a hen's egg was clearly defined about the middle of the upper lobe of the right lung. She suffered from an incessant cough and was restless, but had neither delirium, chills nor sweats, though her forehead was occasionally moist.

Tubercle bacilli and micrococci in sputum; leucocytosis. The necropsy confirmed the diagnosis.

DISCUSSION.

DR. GRIFFITH.—Did I understand that these cases were practically all in the third stage of phthisis?

DR. ADAMS.—Yes.

DR. WENTWORTH.—Were they all cases of general tuberculosis?

DR. ADAMS.—All were pulmonary cases with more or less general tuberculosis.

DR. GRIFFITH.—As I understand it, thesecases are analogous to phthisis in adults, but the range of temperature is greater. I have seen temperature charts a good deal like these in children who did not have tuberculosis.

DR. ADAMS.—The diagnosis of pulmonary tuberculosis, and in some cases of general tuberculosis, was made in all these cases and was confirmed by necropsy. Of course, we frequently see such wide ranges on malarial charts, but they do not continue for two or three weeks, as these did. The question is whether they are cases at all septic in their nature. There was a leucocytosis but no other evidence of sepsis.

DR. HOLT.—It seems to me there must be something else here than the tuberculous process alone. It is well known that we get an exaggeration of all temperature curves in children. I have been accustomed to regard such cases as these as usually due to a complicating infection, generally by the streptococcus. I do not think these children have a general streptococcus septicemia, if that is what Dr. Adams means by sepsis.

A CASE OF ACUTE HEMORRHAGIC NEPHRITIS COMPLICATING INFLUENZA IN AN INFANT OF THIRTEEN MONTHS, WITH AN ANALYSIS OF 40 CASES OF INFLUENZAL NEPHRITIS.

BY D. J. MILTON MILLER, M.D.,

Philadelphia.

At the meeting of the American Pediatric Society, held in Washington, in May, 1900, R. G. Freeman[28] reported a case of acute hemorrhagic nephritis, following influenza, in a child four years old, and in connection therewith analyzed 11 cases of this infrequent complication of influenza. An almost identical case in a much younger child came under the writer's observation during the winter of 1901, and is herewith presented, together with an analysis of 40 cases, which he has been able to collect from the literature.

After the plan and scope of this paper had been determined upon, and the references and other necessary data collected, the writer became acquainted with the article of Dr. Freeman covering almost the same ground; but as the present investigation is based upon more abundant material, and hence more valuable for drawing conclusions, it was decided to continue its preparation according to the original intention, even at the risk of some repetition.

CASE.—Emma, a healthy breast-fed infant of thirteen months, who had never before been ill, was taken sick suddenly on February 19, 1901, with high fever, coryza and cough. The next day vomiting began and was persistent, while the stools were loose and consisted of undigested milk and slightly blood-tinged mucus. The day following (February 21), when

the baby was brought for treatment, she presented the typical features of an ordinary catarrhal influenza, associated with a mild colitis and most harassing cough. The temperature was 102° F., the pulse 120. A fever mixture was prescribed and fractional doses of calomel. On the 22d, following the calomel, there were numerous stools of blood-tinged mucus, the vomiting was less and the temperature had fallen. Amelioration of the catarrhal symptoms followed; but on the 24th of February (sixth day), slight edema of the hands and of the lower limbs, below the knees, with a trifling fulness of the face, made its appearance. The child had passed no urine since early morning, and then but a small quantity, very red in color. The mother thought the baby had headache, as she carried her hand constantly to the head. The initial vomiting, which had somewhat subsided, had recurred with increased severity, but the diarrhea had ceased. By the catheter 30 c.c. of bloody urine was withdrawn, the first passed since early morning. Citrate of potash with warm baths and packs, and sinapisms to the lumbar region were ordered.

An examination of the urine by Dr. A. Hand, Jr., gave the following result: Color, red; strongly acid; specific gravity, ——; albumin, 0.5, by bulk; sugar, negative; bile, negative; deposit, slight; microscope—abundant red blood cells, leucocytes, renal epithelium, many broad and narrow granular casts, numerous blood casts, 3 colloid casts, a few uric acid crystals. Diagnosis: Acute hemorrhagic nephritis.

No increased tension of the peripheral arteries could be detected, but the aortic second sound seemed possibly accentuated. By the 27th the edema was scarcely perceptible, and the urine was passed more freely, about four ounces in twenty-four hours; 19 c.c. of a light red urine were withdrawn by catheter and presented on analysis the same characters as on the 24th, except that the blood cells and various casts were more numerous, while no colloid casts could be discovered. The vomiting had ceased, but the cough was severe and paroxysmal. Examination of the chest was negative. The temperature was 99 4-5°, the pulse 116. The hot packs and baths had induced profuse sweating. Antipyrin was prescribed for the cough, while to the citrate small doses of the bitartrate of potash were added, with a daily bath and laxative dose of magnesia.

From the 27th of February to March 7th the patient slowly

improved; the kidneys acted more freely, but the urine remained slightly blood-tinged. Dr. Hand's analysis of a catheterized specimen on March 4th, showed numerous blood cells and leucocytes, and a few granular and hyaline casts. A week later Dr. A. A. Ghriskey found the urine to have a specific gravity of 1008 (estimated; quantity, 25 c.c.), many blood, epithelial and granular casts, and a few blood cells and leucocytes. From this time there was a gradual improvement, but the urine did not entirely clear up until March 31st (thirty-sixth day of the nephritis). One month later the infant caught a severe cold, with a return of the paroxysmal cough. Simultaneously, some urine of a very slight red color was voided containing a few red blood and epithelial cells, but no casts. This condition had entirely disappeared in a week, and the infant appeared to be in excellent health. After March 20th the treatment consisted in the administration of small doses of Basham's mixture and Fowler's solution. Subsequent to the initial fever, the temperature never rose above 100.5° F., usually ranging between 99° and 100°.

Although nephritis is well-recognized as one of the occasional complications of influenza, it is, according to statistics and the observation of experienced clinicians, of infrequent occurrence. Observers, however, differ as to the frequency of the appearance; thus, in the German collection investigation,[31] it occurred 144 times in 3,185 cases (4.5 per cent.); Anton,[32] of Wurzburg, found it in 2 per cent. of 100 cases; Gmeiner[33] in 1 per cent.; Guttmann[11] 4 times in 262 cases (1.52 per cent.); and H. S. Anders[18] twice in 128 cases (1.56 per cent.). On the other hand, in the German army, only 10 cases of severe kidney inflammation were observed[33] among 55,263 (.018 per cent.) cases of influenza, while O. Leichtenstern[34, 35] saw but 2 instances of nephritis in 439 cases (.45 per cent.), but believes it to be much more frequent, as after the epidemic of 1889 and 1890 much nephritis was seen, in which nothing beyond the preceding influenza could be discovered.

In children nephritis as a complication of influenza is even less frequently encountered. No mention whatever of it is made by Monti,[36] Ashby and Wright[37] and T. M. Rotch,[38] while C. W. Earle,[39] Holt,[40] Unger[41] and Vogel[42] merely refer to its possibility. Baginsky,[43] C. Gerhardt[44] and Henoch[45] also speak of a true nephritis, although the latter had never seen a case,

In the German collective investigation only once was an accompanying nephritis reported in a child.[42] H. Gillet[44] refers to it as a serious sequel, usually attended by hematuria. A. D'Espine and C. Picot,[46] however, have observed it only in benign forms. On the other hand, Fürst[48] observed it 4 times (7.6 per cent.) in 52 cases of influenza in children, and A. Schlossman[46] in 3 per cent. of his cases.

Albuminuria, without the occurrence of true nephritis, is much more common. Senator[28] met with it 18 times in 52, and Brandes[28] 23 times in 29 cases; Anton[30,53] in 5 per cent., Krehl[28] in 3.5 per cent. and Teissier[54] in 50 per cent. of their cases. In 100 children over two years of age Schlossman[46] found albuminuria, and Baginsky[41] and Holt[28] state that it is present in all severe cases.

Hematuria alone is not unusual. This condition was noticed by Curtin and Watson[49] in a considerable number of cases; it was accompanied sometimes by lumbar pains and scanty and dark urine. Instances have also been reported by Krannhals,[52] Drew,[50] Köppen,[13] Poore,[55] Fräntzel[53] and others.[54] Potain[51] remarks that the nephritis of influenza is sometimes preceded by hematuria. No especial reference to its frequency in early life could be found, but in 5 out of 7 cases it occurred under twenty years of age.

Of 70 cases of nephritis complicating influenza, both in adults and children, which a careful search of the literature has discovered, only 40, including the writer's own case, were accompanied by sufficient clinical histories to form a basis for analysis. A brief *résumé* of these cases will be found at the close of this paper.

SEX.—Twenty-six of the 40 cases were in males and 13 in females; in one the sex was not mentioned.

AGE.—The oldest patient was sixty-five years (2 at sixty-five and 1 at sixty), and the youngest five months, of age; 25, or 64.1 per cent., occurred under the age of twenty-five years; 18, or 46.1 per cent., under sixteen, and ten, or 25.6 per cent., under twelve years. Under two years only 2 cases were reported, and those within the first year of life. Thus more than half of the cases were in young adults or children.

TIME OF ONSET.—The statements of Tuvache[56] and Lamarque[57] that the nephritis may come on during the course of the influenza, or insidiously, after the acute symptoms have

passed, are corroborated by this analysis, as the time of onset in 21 of 38 cases was within eight days of the commencement of the influenza, or 56.1 per cent., while 10, or 26.3 per cent., developed between the eighth and thirtieth day; the longest period being sixty-three days, the shortest three, and the average nineteen, days.

TYPE OF THE NEPHRITIS.—Some difference exists as to the clinical features of the nephritis. The ordinary type, according to Potain,[51] is an albuminuria with casts and more or less edema. Fiessinger[15] states that the nephritis may manifest itself in three ways: As a passing glomerulitis, with transitory albuminuria; as an acute hemorrhagic nephritis, without edema, and as acute Bright's disease, the latter being the most common. As noted above, Potain[51] says that the nephritis is sometimes preceded by hematuria, and Tuvache[55] that it may be hemorrhagic; yet in Freeman's[28] analysis it was of the hemorrhagic type in 63 per cent. of the cases. The records on which this study is based show the same preponderance of this form, as blood was present in the urine of 27 of the 40 cases (67.5 per cent.), and in 21 of these, or 52.5 per cent., was in quantities sufficient to warrant the designation "hemorrhagic." Of these 21, 15 were under twenty-five (71.4 per cent.), and 11 (52.3 per cent.) under eighteen years of age, indicating that this type of nephritis is peculiar to young adults and children.

CASTS.—The presence of casts was noted in 34 cases (85 per cent.). In 8 their character was not mentioned; in 6 they were hyaline and epithelial alone, and in 20 hyaline granular and blood. In the writer's case, in addition to the other casts, 3 colloid casts were seen at the first examination only. Such casts do not necessarily indicate a chronic nephritis, much less amyloid degeneration. They are generally believed to be associated with serious lesions, resulting from degeneration of the renal epithelium,[56] and this would explain their presence in the case in question, which, from the intensity of the renal symptoms, was evidently due to a severe infection, while the complete recovery showed that the process was an acute one.*

ALBUMIN.—This was present in 39 of the 40 cases. The quantity was not stated in 9; in 16 it was in large, and in 14 in small, amount.

* According to C. W. Purdy[61] these casts are found in all forms of nephritis.

QUANTITY OF URINE.—No reference as to this point was made in 14 cases. Of the remaining 26, it was scanty or diminished in 20, free in 5 and in 1 there was complete anuria.

EDEMA.—This symptom was observed in 19, or in less than half (47.5 per cent.) of the cases; in 4 of these it was of trifling character, while about one-half (47.6 per cent.) of the hemorrhagic cases were associated with edema.

FEVER.—The onset of the nephritis is usually accompanied by some elevation of temperature. Fever was mentioned in 18 cases; in 3 of these it was over 104° F., in 6 between 101° and 103°, in 3 between 99° and 100°, in 1 between 96° and 97° and in 5 the degree of temperature was not given.

Failure to mention this symptom in the remaining 22 does not indicate its absence, as it is probable that most cases are attended with some rise of temperature, although afebrile cases are not infrequent.

DURATION OF THE NEPHRITIS.—This was stated definitely in 28 cases. In the longest it was nine and five months respectively, both resulting in death; in another, which recovered, it was four and a half months. The shortest cases lasted six days (3 cases), the average for the remaining 25 being nineteen and six-tenth days. In 84 per cent. the duration was under thirty days, in 72 per cent. under three weeks.

TERMINATION.—Recovery is the usual result in the nephritis of influenza (Lamarque,[51] A. Jacobi[47]), although Tuvache[56] states that the prognosis is grave. Occasionally it passes over into the subacute or chronic form (F. Stricker,[51] Potain,[51] Lamarque,[51] Jacobi,[47] Tuvache,[56] Curtin and Watson[49]), or it may terminate fatally. Of the 40 cases, 29 (72.5 per cent.) recovered, 7 died (17.5 per cent.) and 4 became subacute or chronic (10 per cent.); 2 of the cases died of chronic nephritis at nine and five months respectively.

INFLUENCE OF PREVIOUS ATTACKS OF NEPHRITIS.—When influenza attacks a person whose kidneys are already diseased, the prognosis is grave, as it is apt to induce a serious and fatal lesion (Potain[51]). Tuvache[56] saw a case of this nature. V. Budde,[58] on the other hand, observed 2 patients with chronic parenchymatous nephritis, in which, beyond a temporary increase in the albumin and casts, a severe influenza appeared to have no untoward effect.

PATHOLOGY.—Autopsies were made in 4 of the cases. In 1

(Leyden's[13]), a female, twenty-five years old, the kidneys were found large and congested, and microscopically showed a glomerular or capsular nephritis. In the second case (Mosler's[14]), a male, seventeen years old, the autopsy revealed, in addition to other lesions, a hemorrhagic, parenchymatous, interstitial and glomerular nephritis. "The epithelium of the convoluted tubes showed all degrees of degeneration from cloudiness to complete fatty metamorphosis. In places, proliferation of the capsular epithelium, and masses of cells between the glomerulus and capsule; the tubules filled with blood-cells, and areas of small-celled infiltration, especially in the neighborhood of the affected capsules." In the third case (Zahorskey's[20]), an infant of five months, the kidney was softened and congested, numerous small hemorrhages were scattered throughout its substance, with hemorrhages into, and albuminous degeneration of, the uriniferous tubules. In the fourth case (Bonnelière's[25]), no lesion beyond renal engorgement was mentioned. The anatomo-pathological lesions of influenzal nephritis are, according to Potain,[51] difficult to determine. In addition to glomerular and parenchymatous nephritis, Du Ponchell[51] reported a case of interstitial nephritis, Beneke[56] 2 cases in which extensive necrotic processes had occurred and Tuvache[56] a case of mixed nephritis. The lesions, indeed, are those of an infectious or toxic nephritis, whose type is the scarlatiniform, the glomerulo-nephritis of Klebs (Tuvache[56]).

From this study the following conclusions seem warranted:

Nephritis is a rare complication of influenza, occurring chiefly in young adults and children, and in infancy is almost unknown. Albuminuria is more common, and is probably present in all severe cases. Hematuria alone is not infrequent, and, like the nephritis, is seen oftenest in early life. The sexes are affected equally. The nephritis may appear early in the influenza, or at varying periods after the acute symptoms have subsided. It is usually an early complication, occurring in one-half the cases within eight, and in two-thirds of the cases within twenty-one days of the commencement of the influenza. The clinical type varies; it may be that of an ordinary acute nephritis, but in the majority of cases is of the hemorrhagic type, this form being especially frequent in young adults and children. Edema is absent in more than half of the cases, and is apt to be slight. The onset is usually attended

with fever. The nephritis is of short duration, generally lasting under three weeks. The prognosis is good, recovery being the rule; although a small number of cases passes into the chronic or subacute stage. When influenza attacks those whose kidneys are already diseased, it is apt to be serious and fatal. From the meagre pathological reports the lesions appear to be those of an infectious or toxic nephritis, often taking the form of a glomerulo-nephritis.

RÉSUMÉ OF REPORTED CASES OF INFLUENZAL NEPHRITIS.

I. A. A. HOEHLING.[1]—Male, twenty-five years; gonorrhea, February 11, 1890; on February 25th, influenza; March 9th, urethral discharge almost ceased; March 11th, chill, lumbar pains, temperature 100°–103° F.; March 17th (twentieth day), blood casts and albumin in urine; April 27th (seventeenth day of nephritis), recovery; nephritis lasted seventeen days.

II. IBID.—Male, influenza in December, 1889; nephritis shortly after; February 28, 1889, albumin ¼ per cent., hyaline and granular casts, which persisted at last examination, May 17th.

III.—LESNÉ.[2]—Male, eleven years; February 28, 1895, severe influenza, temperature 40° C.; March 4th (fifth day), 500 gms. of dark urine, containing blood and albumin (notable amounts), temperature 39.8°, 38.4° C.; March 7th, edema of the face, 250 gms. urine; March 9th, acute rheumatism and pleurisy; old mitral insufficiency; rheumatism lasted until March 15th, when hematuria returned and albumin; March 16th, recovery; nephritis lasted thirteen days.

IV. DANFORTH.[3]—Male, sixty-five years; one week after moderate attack of influenza, relapse, with intermittent pyrexia; urine, twenty to twenty-four ozs., specific gravity, 1024-30, much albumin, few red blood-cells and leucocytes, mucin and hyaline casts; in four weeks, recovery; nephritis lasted twenty-eight days.

V. G. BAUMGARTEN.[4]—Female, forty years; December 3d, moderate influenza; last days of December (twenty-seventh day), headache, edema and slight fever; January 2d, albumin and few casts: January 8th, urine bloody and blood in varying amounts till January 29; casts were various; specific gravity, 1024-22-17, leucocytes and many red blood-corpuscles, albumin 1-10 to 1-3; nephritis lasted four and a half months.

VI. IBID.—Male, forty-four years; influenza November 9th; never fully recovered strength; December 14th (thirty-sixth day), headache, malaise, leg-pains, cough and albuminuria; fourteen days later, edema of legs; albumin ⅝ per cent., hyaline and granular casts; died from hydropericardium; never had nephritis before.

VII. IBID.—Male, sixty-five years; influenza in March; March 30th, sudden increase of fever and severe abdominal pain; April 6th (thirty-sixth day), urine scanty, specific gravity, 1022, 1 per cent. albumin, granular and hyaline casts; traces of albumin and casts persisted until June 3d; recovery; duration of nephritis, fifty-eight days; three years later in good health.

VIII. IBID.—Female, eighteen years; scarlet fever at thirteen years, kidney not affected then; severe influenza in winter of 1892-93; never fully recovered health; April 14th, 1 per cent. albumin, leucocytes, red blood-corpuscles, hyaline, granular and epithelial casts; condition persisted until January, 1895, when death in uremic coma; edema of legs, ankles and face; duration of nephritis, nine months.

IX. IBID.—Female, nineteen years; scarlet fever in childhood; five years ago mild diphtheria; August, 1894, influenza followed by otitis media; continued ill-health, sick headache and vomiting; in March, 1895, albuminuric retinitis; March 25, 1895, second attack of influenza; urine contained ⅔ per cent. albumin, specific gravity, 1025, hyaline casts; albumin still present at date of report (May, 1895).

X. IBID.—Female, forty-three years; moderately severe influenza, December 12, 1894; December 26th (fifteenth day), scanty urine, ½ per cent. albumin, hyaline and granular casts, white and red blood-cells; edema of legs and hands; recovery, February 3d; nephritis lasted forty days.

XI. IBID.—Male, twenty-seven years; influenza May 14, 1895; severe, high temperature, vomiting, backache; May 18th, 200 c.c. of bloody urine (*i.e.*, fifth day), ½ per cent. albumin, specific gravity, 1018, numerous red blood-cells and casts, epithelial and granular casts; temperature 101° to 103° F.; May 19th–20th, scanty and bloody urine (300 c.c.) again; vomiting occasional; gradual improvement, which continued to date of report (May, 1895); probable recovery.

XII. J. W. FRASER.[8]—Male, nine years; March 2d, influenza of

ordinary type; March 7th (sixth day), scanty bloody urine, albumin 0.2 per cent., blood and granular casts, renal epithelium; temperature 101° F.; trifling edema of ankles; March 10th, specific gravity, 1026, hyaline casts, trace of albumin; March 28, recovery; nephritis lasted twenty-two days.

XIII. E. MANSEL-SYMPSON.[6]—Male, eleven years; influenza beginning of March (severe); lasted two weeks, left patient debilitated; March 16th (sixteenth day), sudden, severe vomiting, headache and lumbar pains; temperature 100.1° F.; ankles edematous; one pint urine (twenty-four hours), specific gravity 1015, hyaline casts and blood-cells, albumin, ⅜; April 1st, only trace of albumin; recovery; nephritis lasted a "few weeks."

XIV. W. B. RUSSELL.[7]—Young woman; severe influenza; backache, abdominal pain and vomiting; with these symptoms, scanty urine, dark porter color, some red blood-cells, numerous epithelial and blood casts, abundant albumin; recovery; nephritis lasted "some weeks."

XV. IBID.—Young woman; influenza with severe sore throat; temperature, 102° F.; shortly after onset scanty and very bloody urine ("looked like pure blood"), numerous blood casts and red blood-cells; extreme cardiac weakness and vomiting; fever; epithelial replaced blood casts in a few days; albumin; recovery; nephritis lasted a "few weeks."

XVI. N. BOCK.[8]—Female, thirty-two years; beginning of December, influenza; convalescent in ten days, but debilitated; December 18th (eighteenth day), dark urine containing metahemoglobin and numerous red blood-corpuscles; left lumbar pain (left kidney larger than right); urine scanty (300-500 c.c.), numerous epithelial and blood casts, small amount of albumin; hematuria lasted until all trace of renal disease had disappeared; recovery complete in three weeks.

XVII. E. A. PIGGOTT.[9]—Male, forty-two years; June 9th, severe influenza, temperature 104° F.; June 11th (third day), dark, smoky urine, numerous red blood-cells, large amount of albumin, epithelial casts; pulse and temperature continued high; June 13th, subsidence of acute symptoms; slow convalescence; recovery.

XVIII. FR. MOSLER.[10]—Male, seventeen years; four weeks after severe influenza, intense lumbar pains, high fever (40° C.), sopor, apathy, irregular pupils; scanty, bloody urine; later

complete anuria, coma and death on thirty-sixth day; autopsy: gastroenteritis, pleurisy, hypostatic pneumonia and bronchitis, pericarditis and parenchymatous nephritis, with numerous punctate hemorrhages; microscopic diagnosis: nephritis hemorrhag. parenchym. et interstitialis; glomerulo-nephritis; nephritis lasted eight days.

XIX. F. GUTTMANN.[11]—Male, eighteen and a half years; early in influenza when bronchitis was severe, with free urinary flow (1075 c.c.), albuminuria (1½ pro mille—Esbach—to ¼ pro mille), specific gravity, 1017-18; red and white blood-corpuscles and hyaline and granular casts; recovery.

XX. IBID.—Male, fifty-four years; during an influenzal pneumonia, albuminuria (1 pro mille to 25 pro mille), and hyaline and granular casts; urine, 750 to 1050 c.c. daily; specific gravity, 1014-16; recovery; nephritis lasted seven days.

XXI. E. LEYDEN.[12]—Female, twenty-five years; influenza in beginning of December; remained weak; beginning of January (thirty-fifth day), eight days of violent vomiting; urine dark, scanty, 200 c.c. daily, contained blood and abundant albumin; otitis present and high grade of anemia; death; autopsy: kidneys large and congested, and microscopically showed glomerular or capsular nephritis; nephritis lasted thirty days.

XXII. A. KÖPPEN.[13]—Male, thirty years; severe influenza on March 13; temperature 38.8° C.; vomiting, intense back and head pains; March 15 (third day), scanty, dark urine, specific gravity, 1024, albumin, ½ per cent., large number red blood-cells and leucocytes, former in heaps and casts; amount of urine, 250 c.c.; hematuria persisted till March 23; recovery; nephritis lasted eight days.

XXIII. BAUDET.[14]—Child, nine years; ordinary influenza; on twelfth day edema of malleoli and face and lumbar pains; urine contained albumin and casts; recovery; nephritis lasted ten days.

XXIV. CH. FIESSINGER.[15]—Male, sixteen years; March 18th, severe influenza, chill, headache; temperature 40.7° C.; cough, diarrhea and vomiting; March 21 (fourth day), urine bloody, 1 litre in amount, epithelial casts, numerous red blood-cells and albumin; recovery on March 25th; nephritis lasted six days.

XXV. IBID.—Female, three years; influenza on December 20th; temperature 40.8° C.; bronchitis; January 11th (twenty-third day), lumbar pains and edema of feet and face; large

amount of albumin, epithelial casts; temperature 37.8°–9° C.; recovery, January 28; nephritis lasted seventeen days.

XXVI. IBID.—Male, eight years; influenza with angina on February 9th; temperature 39.8° C.; February 13th (fifth day), edema of the face; albumin and epithelial casts; February 26th, urine free; recovery; nephritis lasted fourteen days.

The same author, in another paper,[16] describes an epidemic of acute nephritis occurring in a small village. Although influenza was epidemic at the time, and notwithstanding that 9 of the 14 cases were immediately preceded by influenza, he did not consider that the latter bore any etiological relation to the nephritis, because the 5 remaining cases were not preceded by that disease. Three of his cases, however, appeared to be so closely connected with an attack of influenza that they are included in this *résumé*.

XXVII. IBID.[16]—Female, seventeen years; influenza end of January; eight days after recovery, February 17th, headache and anorexia, vomiting, edema; February 21, urine scanty, contained blood, albumin and epithelial casts; few days later granular casts in large numbers, temperature 40° C.; February 27, death; nephritis lasted eleven days.

XXVIII. IBID.—Male, sixteen years; middle of February influenza; on eighth day from onset of influenza, edema of the face; five days later granular and epithelial casts; on ninth day of nephritis, uremic convulsions, scanty urine and 4 per cent. albumin; complete recovery; nephritis lasted nineteen days.

XXIX. IBID.—Male, twenty-one years; influenza in middle of February; on eighth day (February 22d) abdominal pain and edema of legs; urine contained traces of albumin and casts; complete recovery by March 1st; nephritis lasted seven days.

XXX. WEICHSELBAUM.[17]—Young girl; severe influenza at Christmas; January 11th (seventeenth day), high fever, intense lumbar and leg pains; urine rich in albumin; recovery; author regarded it as nephritis complicating influenza.

XXXI. H. S. ANDERS.[18]—Male, thirty-eight years; alcoholic and syphilitic; characteristic influenza; in a few days edema; copious albumin (⅓); tube casts, epithelial and granular; death in uremic coma.

XXXII. IBID.—Male, eighteen years; influenza; in a few days edema; albumin (⅓) and granular and epithelial casts; extreme anasarca; recovery.

XXXIII. VIGNEROT.[19]—Male, thirty-three years; January 15th, severe influenza; chills, fever, intense lumbar pains; convalescent in eight days; March 10th (forty-sixth day after recovery from grippe), edema of legs, urine 1½ litres daily, 5 gms. albumin per litre; death from uremia May 2d; nephritis lasted fifty-four days.

XXXIV. IBID.—Male, thirty-one years; influenza in February; resumed work in eight days, but did not fully recover; in May, urine dark red, scanty; vomiting and edema, lumbar pains; urine 1 litre in twenty-four hours, considerable albumin; temperature 36° C.; in September uremic convulsions; death September 22d; duration of nephritis, five months.

XXXV. JOHN ZAHORSKEY.[20]—Male, five months, previously apparently healthy; attacked with influenza January, 1900; on third day cerebral symptoms, slight cervical opisthotonos, small pupils, coma; persistent cough, chest examination negative; urine not examined and hematuria not observed, although may have been present; death in one week from onset of influenza. Autopsy: Nothing of note in brain or thorax; right kidney softened; small hemorrhages scattered throughout substance; microscope showed hemorrhagic areas and albuminous degeneration of tubules; left kidney normal; cultures from diseased kidney remained normal; nephritis lasted eight days.

XXXVI. J. G. EDGREN.[21]—Male, thirty-six years; influenza December 11th; recovery, but remained debilitated; February 18th (sixty-ninth day), albumin discovered; urine, 1,100 c.c. daily; 0.1 per cent. albumin, hyaline casts, leucocytes and renal epithelium; condition continued until discharge, March 13, 1890.

XXXVII. ÉMILE BONNELIÈRE.[22]—Female, sixty years; influenza January 19th; severe pains in loins; temperature 39.5° C.; marked bronchitis and diarrhea; in a few days traces of albumin in dark red urine, casts and much blood; symptoms increased until death on January 30th; nephritis lasted thirteen days. Autopsy: Kidneys gorged with blood.

XXXVIII. IBID.—Male, seventeen years; February 13th influenza of severe type; from onset severe lumbar pains and dark urine; temperature 39° C.; typhoid state; pulmonary congestion and pericarditis; slight edema of face and malleoli; defective sight; 1 grm. albumin per litre urine, numerous red blood-cells

and casts; hematuria lasted until March 5th; recovery; nephritis lasted twenty-five days.

XXXIX. R. G. FREEMAN.[58]—Male, four years; January 1, 1899, ordinary influenza with moderate fever, prostration, earache without discharge; January 31st (thirtieth day), temperature 102.5° F.; highest temperature 105°, on February 5th; temperature diminished, and on February 9th varied between 100° and 101°; normal on the 10th; February 6th passed red urine, containing on the 9th considerable blood, 2 ounces in twenty-four hours; later 6-14 ounces daily; albumin, 5 per cent. (bulk), hyaline and blood casts; specific gravity, 1022; blood persisted five days, casts ten days; no edema; child well one year after; nephritis lasted ten days.

In addition to the above, the following cases of influenzal nephritis are reported with clinical histories too meagre for analysis: Diaro,[57] 2 cases, one in a female, eighteen years, ending in death; a second in a male, forty-four years, with death on fifteenth day. Anton,[59] 2 cases, one ending in recovery on the second day; the other, with abundant albumin and casts, also recovered after a short illness. Strümpell,[58] 2 cases, one of pronounced hemorrhagic character, which disappeared in a few days; another, more severe and prolonged, in an influenza empyema. Fussel,[60] 2 cases of post-influenzal parenchymatous nephritis. Teissier,[54] a case in a female of eighteen years, terminating fatally. Shattuck,[56] Chostowski,[55] Umpfenbach[60] and J. I. Bolton[6] have also reported cases.

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AMAUROTIC FAMILY IDIOCY.

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Thus far our researches of cases of this interesting disease have failed to find more than 36 recorded. In 1889 Sachs collected 29 cases. Since then Hirsch, Peterson, Patrick, Kuh and Welt-Kakels have reported 1 each and Claiborne claims to have seen 5.

REPORT OF CASE.—Rosa F. aet two years, of Hebrew parentage was referred to me June 10, 1900, as a case of rachitic malnutrition.

FAMILY HISTORY is negative except for fatal tuberculosis in a paternal uncle. (Later I learned that the father's cousin's only child, J. S., died in convulsions at twenty months, blind, idiotic and never having been able to stand or sit erect. Concerning this latter child the attending physician, Dr. Ginsburg, states that he first noticed evidence of blindness a little before age of six weeks.)

The parents of the patient are living and in good health; well-to-do tradespeople, able and anxious to give every possible advantage in the care of their child. There is no consanguinity of parents and no history of syphilis. An older child died at 7½ months of cholera infantum. A younger child, now six weeks old seems normal.

PERSONAL HISTORY.—Gestation and birth were normal. Initial weight 9 lbs. Nursed 15 months and then was weaned because of pregnancy of mother. Seemed well in every respect until six months of age when she had an attack of bronchitis. From this she made a good recovery, but soon after suffered from a second cold from which convalescence was somewhat tardy. Has never had any of the exanthemata. Eruption of first tooth occurred at one year of age. It is stated that at the usual time (about the ninth month) the patient was able to stand with support. From this time on the child showed progressive

muscular debility with increasing disinclination to use limbs. From earliest infancy she was easily startled. Appetite was good, although solids were swallowed with difficulty. Always constipated. Blindness has gradually developed.

PRESENT CONDITION.—June 10, 1900.—A plump child of average size, with some evidence of atrophy of lower extremities; anemic. Head measures 19 inches in circumference. Expression is blank except when the face shares in general convulsion. Convergent strabismus is present. Pupils are unequal in size, the right being twice the size of the left. There is no response to light. All tests fail to give any evidence of vision and the child undoubtedly is totally blind. There is a high arched palate. She is unable to hold up her head or to use her limbs. The mother says she lies on either side, but whenever seen the face is drawn to the right. There is spasticity of both upper and lower limbs, most marked in right arm. "Claw hands."

Reflexes are enormously exaggerated. A striking feature is the sensitiveness to sound and touch. The slightest noise or the least jar brings on a spasm, during which the eyes waver vertically *i.e.*, nystagmus, the arms are rotated internally and adducted, there is frothing at the mouth and the whole body is slightly involved. These spasms last from fifteen to sixty seconds and are followed by moaning. They are repeated every time she is disturbed. Temperature normal, pulse rather rapid.

I saw the child several times, both at my office and at the clinic. At the first visit a positive diagnosis was not made but the girl was presented at the clinic as a possible case of cerebellar tumor of tubercular character. She was referred to Dr. C. D. Wescott for examination of fundus. His report cleared the diagnosis. It is as follows:

"The case of Rosa F., whose eyes we examined June 26, 1900. The examination was quite difficult for the reason that the child's head had to be held and she was extremely sensitive to external impressions; furthermore, she had quite characteristic lateral nystagmus, which also increased the difficulty of getting more than a momentary view of any part of the fundus. It was possible to make out, however, that the discs were normal and the choroidal rings distinct. The choroid, so far as could be seen, seemed perfectly normal. The feature of the eye ground, which at once attracted attention, however, was a

large, dark reddish-brown disc, occupying the situation of the macula and surrounded by a larger whitish zone about twice or two and a half times the diameter of the optic disc. It was possibly a trifle longer horizontally than vertically; apparently denser near the centre immediately around the dark colored disc and gradually shading off into the normal red of the surrounding eye ground. The brown disc at the macula was regular in outline and decidedly not as red as the spot which we see in embolism of the central artery of the retina. As pointed out by Beard, the whitish zone about the central dark spot was nebulous, rather than cloudy, and denser immediately about the dark spot. The retinal vessels which entered the area, could be seen through it."

After several visits the case was lost sight of because the family moved to the country for the summer. The subsequent history was obtained from the mother and shows nothing eventful except the increasing difficulty in swallowing, inanition and death during a convulsion on August 1, 1900. No autopsy was allowed.

Amaurotic family idiocy is a name suggested by Sachs for a disorder which begins during the first six months of life in apparently normal infants, without history of complicated labor and terminating in death before the third year. (One exception is recorded by Peterson. He reports the child living at the age of five and one half years, blind and idiotic. Two other members of the family succumbed to this disease at seven and ten months respectively.)

As now understood the group of symptoms is sufficiently constant and peculiar to render diagnosis easy and unmistakable. Of these amaurosis is always present, and the ocular findings as accurately described for the first time by Warren Tay in 1881, have come to be regarded as pathognomic of this disease. The foregoing presupposes a complete development of the disorder and presentation of all the clinical findings.

Diagnosis without the characteristic changes of the macula lutea the disorder might be mistaken for rachitic pseudo-paralysis, as was the case with Jacobi and my predecessor. Cerebellar tumor which actually appeared as a complication in the case reported by Claiborne; tubercular marasmus, spastic diplegia or congenital blindness in an idiot. All of these have probably served as diagnoses in many unrecognized cases of

amaurotic family idiocy. So, too, in the early stage, with the most careful ophthalmoscopic examination, difficulty in diagnosis may obtain because of the lack of full development of ocular changes, as in Koller's and Peterson's cases. The difficulty of making a satisfactory ophthalmoscopic examination, the evident lack of mental development and the welcomed early death all tend to discourage a painstaking diagnosis and induce the writer to believe this disease is not so rare as the limited number of cases recorded would suggest.

Of the symptoms four are constant, viz.: idiocy, characteristic changes in macula lutea, paralysis, and early death. Of the other symptoms recorded there may or may not be nystagmus, strabismus, spasticity, flaccidity, convulsions, tremors, exaggerated, normal or subnormal reflexes, dysphagia, hyperacusis and hyperesthesia. Semitic parentage has been noted in nearly every case.

Although most of the reports from Tay down recognize a suggestive family character to this disorder, many exceptions are observed, as in my own case—but one of a family of three being affected.

Since the prognosis is invariably fatal and all therapeutic measures are admittedly hopeless, our interest mainly centres on etiology with a view to prophylaxis.

Autopsies, though few in number, seem to establish the fact of a progressive degeneration of the cellular elements of the entire nevous system, though whether primary or secondary still seems a matter of dispute. No vascular or inflammatory changes have ever been found. Sachs' change in name from agenesis corticalis is suggestive of his theory of its etiology—to amaurotic family idiocy—a clinical classification—expresses the absence of definite knowledge at present. Hirsch's theory, somewhat favored by Jacobi, of an infection, possibly derived from the mother's milk, if susceptible of demonstration, promises an easy solution to the question of prophylaxis in artificial feeding, as these children seem normal at birth. Opposed to this theory of some toxin in mother's milk is the fact, brought out by Sachs in the discussion, that two of his cases were not breast-fed.

Report of this case has been delayed with the hope that observation of the surviving child, as well as of a cousin's child, would add to the interest. The remaining child, now nearly

one year old shows no marked departure from the normal, except a tendency to rachitic development, with a history of convulsions during a febrile attack at six months. This was reported as due to indigestion.

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DISCUSSION.

DR. MORSE.—I merely wish to say that I agree with the reader of the paper that these cases must be more numerous than we should judge from the number that have been reported. I myself have seen three typical cases with the characteristic eye grounds; two were of Jewish parentage and one of American. They all died at an early age.

HIGH TEMPERATURE OF THREE MONTHS' DURATION, WITH DOUBLE SACCULATED EMPYEMA, SITUATED ON ONE SIDE ON THE DIAPHRAGMATIC SURFACE OF THE LUNG.

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Few conditions are more puzzling than cases of prolonged high temperature when it is not accompanied either by physical signs or rational symptoms which are proportionate to the degree of fever. The case, the temperature chart of which is presented, was one of the most obscure that has come under my observation. It was peculiar not only for the duration, but the wide fluctuations of the temperature, for which no adequate cause could be found, and no diagnosis that was satisfactory was reached during life.

The patient, a boy two and a half years old, was admitted to the New York Foundling Hospital, on January 10, 1901, and was acutely ill until his death on May 2d, a period of nearly four months. The complete chart is not given on account of its size, but the temperature curve for the periods omitted, differed in no important way from that which immediately preceded or followed. He came under my personal observation three weeks before his death, having previously been under the care of my colleagues, Drs. J. J. Reid and R. G. Freeman.

On admission the general condition was good, and the child showed no evidence of any serious illness, except a moderate diarrhea which the history stated had existed for some time. For the first two weeks of observation his temperature ranged from 98° to 100°, except upon a single day; his stools

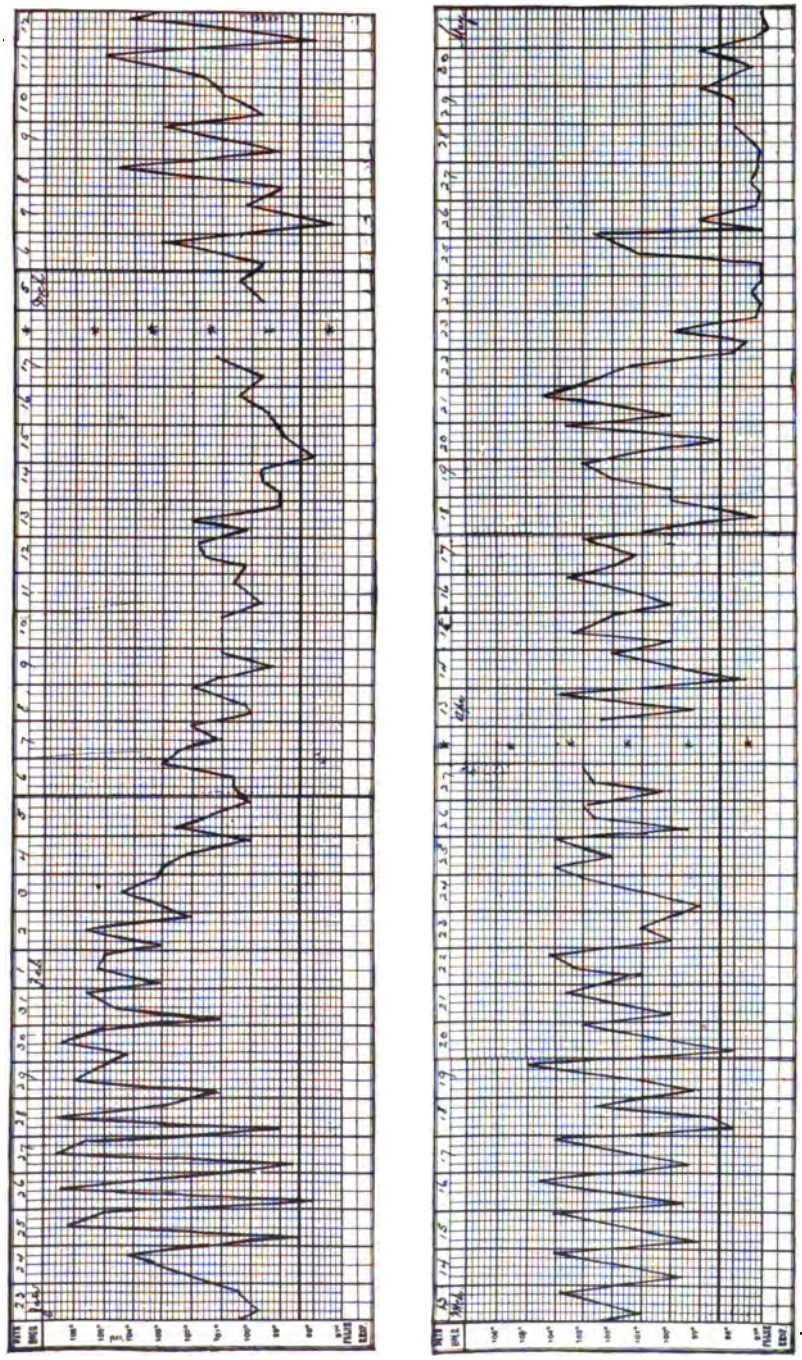
were somewhat indigested, were rather frequent and at times contained mucus. He did not, however, appear seriously ill.

During the third week was witnessed a sharp rise in the temperature, which was characterized by wide fluctuations between 98° and 106.2° F. As the spleen was considerably enlarged, although no malarial organisms could be found in the blood, on repeated examinations, quinine was administered in full doses both by mouth and hypodermically, but without any perceptible effect either on the temperature or the general symptoms. It was not until the high temperature had lasted for four or five days that any signs of disease were discovered in the lungs, and then only a few rāles were present. Not until the ninth day, February 3d, were there any signs of consolidation detected; these appeared in the right upper lobe which became later almost completely solidified.

These signs greatly diminished in the course of the next few days as the temperature declined; but they did not entirely disappear and nearly always thereafter were to be heard a few coarse rāles, occasional friction sounds, and rude breathing, while the percussion note remained slightly dull. The intestinal condition did not improve during this acute attack, nor was it distinctly worse. There were from five to ten passages a day, mostly small, usually loose and sometimes foul. The symptoms continued without special change throughout the month of March, the patient still very anemic and steadily losing weight. Widal tests were made with negative results. A leucocyte count made March 20th, showed 28,000.

On April 1st, there was a slight increase in the pulmonary signs, and rāles were now heard over both sides of the chest. On April 12th, at the base of the left lung, near the spine, slight dulness was present over a small area with rather feeble bronchial breathing; the signs upon the right side remaining about as before, viz.: rather feeble breathing, very slight dulness but coarse rāles and friction sounds everywhere.

On April 18th, the leucocyte count showed 10,500 and the signs at the left base were less marked. The sputum was examined for tubercle bacilli with negative results. On April 25th, the leucocyte count showed 12,500. At this time the signs on both sides of the chest were about the same. There



TEMPERATURE RANGE IN CASE REPORTED BY DR. HOLT.

was a moderate dulness on percussion, but no flatness. Breathing sounds were heard all over the chest; the quality was in several places broncho-vesicular, but nowhere bronchial, and at no part of the chest were râles absent. At no time were there any signs of importance anteriorly. The stools continued frequent, ranging from three to eight a day, most of them containing mucus, the character varying with the food taken. The child became extremely anemic and wasted steadily. The intestinal condition seemed quite sufficient to explain the general symptoms, with the exception of the temperature and it was difficult to find any explanation for this and the enlarged spleen, which was satisfactory. There was very little cough, the respirations were slightly accelerated, but scarcely more than would be expected with the temperature. There was no point where the signs seemed sufficient to justify the introduction of a needle. During the last week of his illness, without any assignable cause, the temperature fell to nearly normal, and remained so until death. There was no displacement of the heart at any time, no vomiting, no cerebral symptoms. The weight fell from twenty-six to sixteen pounds.

AUTOPSY.—Body extremely emaciated. There were very extensive adhesions over the greater part of both lungs, so firm that the lung was torn in removing them from the chest, the pleural cavity being nearly obliterated. There were two small sacculated empyemas; the one on the right side being situated on the diaphragmatic surface of the lung near its anterior margin; the one on the left side being near the base of the lung and close to the spine. Each contained from one to two ounces of thick creamy pus which was not putrid. The upper lobe of the right side was partially consolidated, and on section it was grayish in color and showing a good deal of increase in the connective tissue of the lung; a very similar appearance on the left lower lobe, though not nearly so marked. The spleen was three times its normal size but in other respects normal. Throughout the colon were evidences of old inflammation,—very marked thickening and old ulcers undergoing cicatrization, together with some recent congestion.

The other organs were essentially normal.

The anatomical diagnosis was double sacculated empyema, persistent bronchopneumonia, chronic colitis.

REMARKS.—In reviewing the symptoms of this long and obscure case it seems, even in the light of the autopsy, difficult to explain the wide fluctuations in temperature. It seems to have been due to a combination of conditions. The pneumonic process was an acute pneumonia of the right side, which never entirely resolved, but was followed by interstitial pneumonia with an extension, about a month before death, to the left lung. The collection of pus on the diaphragmatic surface of the right lung, of course, could not be reached by a needle and was not recognizable by any physical signs. The sacculated empyema on the left side situated against the spine was almost as inaccessible, as it scarcely came to the surface of the chest at all. The enlargement of the spleen seems to have been due to the venous congestion resulting from the interference with the pulmonary circulation.

Throughout the illness the child gave the impression of one who is very seriously ill. No treatment had any effect upon his symptoms, and while the physical signs varied somewhat from time to time, were so much alike upon both sides that it added greatly to the obscurity of the case, and it made one doubtful how much importance was to be attached to the rather feeble breathing and the very slight dulness which were the only persistent conditions. A certain amount of interstitial pneumonia, with pleuritic adhesions following an acute process, was expected, but the two pockets of pus which were discovered at the autopsy were a surprise.

DISCUSSION.

DR. CHAPIN.—I would like to report a case of persistent high temperature which was extraordinary in many respects. The child was observed by another physician for several weeks. It was then sent to me at the hospital, and I kept it for two months with the temperature going up regularly every fifth day. An examination of the organs and of the blood was very carefully made but no cause was found for the fever. Clinically it looked like malaria, but quinin, Fowler's solution and Warburg's tinc-

ture had no effect upon the temperature. Gradually the child recovered a normal condition.

DR. WILSON.—I should like to make a suggestion with reference to these cases of continued high temperature, that the examination should not be considered complete until a very careful note is made in regard to the heart. An adult case of this kind would suggest the presence of endocarditis, not necessarily malignant.

SUN PLAY-ROOMS ON CITY ROOFS.

BY W. P. NORTHRUP, M.D.,

Professor of Pediatrics, New York University and Bellevue Hospital Medical College, New York.

For fifteen years I have been eyeing the city roofs in New York, thinking how fine the air, how free from dust, how exposed to the sun and wondering why they have never been used for the children's play-houses. People are building tall houses, are beautifying the sky line with towers and spires, and sending their first-born out for recreation in the crowded street, to mingle with unknown children and adults, their nurses hobnobbing with each other, and both with the policeman.

Recently an opportunity was given to me to carry out the idea. Not many months ago a delicate infant, who had been under the care of Dr. Rotch, came to the city to live. She was the only female child for some generations and had devoted parents and grandparents who wished to do everything to improve her vitality.

The house of my patient faces west and is on one of the hills of Madison Avenue. Over the front roof there was no obstruction. There was suited to our purpose a space of 25x40 feet perfectly unobstructed and a grandfather's backing to develop an ideal roof sun play-room. To this problem were set the architects accustomed to similar propositions in the Presbyterian Hospital. The result was satisfactory. First two strong iron girders were laid from side wall to side wall. Upon these rested the weight of the room; then an iron frame was erected, the outlines of the room. The roof was iron with glass side-covering of sheet-iron with figured pattern resembling brick. Inside there was wainscotting of yellow pine; flooring was likewise of yellow pine. From floor to peak of roof was 16 feet. The interior of the room was 20x20 with a vestibule, the latter being over the stairway from the house below. Between the vestibule and large room there was a wicker gate, some heat coming

up from the house through the stairway. In addition there was placed a well fenced-in gas-stove, which can be started at any time to take off the chill.

The room is lighted on three sides and by sky-light; on the north the wall is solid. Three sides are capable of being opened freely. The glass windows are constructed so that they can be swung to any angle and fastened according to the needs of wind or of bad weather. The roof-light can be cut off by curtains and the ventilators can be closed. Heating of the room

INTERIOR OF SUN PLAY-ROOM.

at all seasons is mostly by sun. In cold, bright, sunny days in winter there is no difficulty in keeping the room in fit condition for the child to play in; indeed no one who has not experienced the heat of a sun-room is prepared to realize its *agreeableness.*

Many New York families feel it necessary, under the advice of their physician, to send delicate children to southern climates during the dusty, windy month of March. This is a thing of luxury and it has its objections, for they are removed from the comforts of a home. This spot is above the noise, out of the

dust, free from contact of strangers and stragglers, away from the dangers of trolley-cars and automobiles, to say nothing of the perils of bicycles and roller-skaters.

Since this opportunity to build an ideal sun-room arose, I have had opportunities to develop other places less extensive for getting children out in the sun in winter weather. One patient has built a dining-room extension 30x20 feet. The roof of the dining-room extension is a platform, the floor of tile, and around this platform is a parapet three feet high. Such is this

EXTERIOR OF SUN PLAY-ROOM.

favorable corner that this extension has an exposure getting sun on some part of it at nearly all hours of the day. On another occasion a sick child was believed to have tuberculosis and a temporary sun-room was built in an angle of walls with an awning stretched over it. This was temporary, easily constructed, but served its purpose.

If I have called to the minds of the American Pediatric Society, how the air and sun in crowded New York may be made to warm and cheer new corners for the use of growing

infants, my purpose is accomplished. Let us remember that the modern treatment of tuberculosis teaches us that for promoting active, complete metabolism, cool, fresh air is our best aid.

It is easy to think that, in case of needed rigorous isolation, a patient might easily be treated through any of the contagious diseases without contact with others. Not the least of its uses is the ability to treat whooping-cough in open air free of dust in the worst months of winter.

DISCUSSION.

DR. PUTNAM.—I am glad to find this plan carried out, for it is one I have always wished to see. There would be one difficulty in Boston; that the conductors that take the water from the roofs connect so that at the windows of the French roofs there is frequently a strong odor of sewer gas. One can manage the water conductors of his own house, but it is the conductors from his neighbor's house that would trouble him in Boston. The tall pipes shown in these illustrations are probably the ventilators of soil pipes, but I refer to the water conductors from the gutters of the house.

DR. NORTHRUP.—Our houses are not built in that way. We did fear the effect of gas from the furnace chimneys, but it has given us no trouble.

DR. ADAMS.—In the house next to mine in Washington the attic was converted into a conservatory and the idea struck me as it did Dr. Northrup, that many houses might be utilized with some contrivance of this sort for the use of young children. I would say, however, that the neighbors of these sun-rooms who have to stay in the city after the hot weather begins may suffer from them. The upper part of my house is uninhabitable at that time of the year because of the heat that comes from my neighbor's sun-parlor.

DR. WINTERS.—A friend of mine had some such room constructed on his house but found it impracticable to use it because the children had more colds than usual. At the Society for the Prevention of Cruelty to Children we have such a room properly protected as regards temperature and draughts, but we constantly have cases of tonsillitis that I attribute to the use of this play-room.

DR. ADAMS.—Would you not attribute that to faulty ventilation?

DR. WINTERS.—The ventilation is perfect in every way. It may perhaps be due to exercise or the asphalt floor.

DR. ROTCH.—How does Dr. Northrup keep this room warm?

DR. NORTHRUP.—It is not intended to keep it warm, but a gas-stove is kept there to use if necessary in excessively cold weather.

DR. GRIFFITH.—It seems to me that if one is going to build such a roof garden it should be arranged not for the month of March only. If you have a glass roof the garden will become unbearably hot later in the season. This heat cannot be satisfactorily controlled by the use of awnings and curtains. The roo fshould be opaque, and the windows on each side should extend from the eaves to the floor. This allows the sunshine to strike far into the room at every hour except high noon, and even then it enters well from the south. We have a satisfactory room at our Children's Hospital, and I think the same principle may be equally applied to private houses. This roof garden has been thoroughly tested and has been of the greatest service. All the sashes are removable for use in summer, while on the coldest winter days a small compartment at one end of the garden is heated. Into this a child can go for a time should it be feared that the cold is too great.

DR. NORTHRUP.—A very good roof garden and one which gave me confidence to go ahead with this enterprise is the roof of the Mothers' and Babies' Hospital. At the Foundling Hospital we have been acting on the principle of getting the children out of doors as much as possible, and they take on a new lease of life. They stand their illness much better, are more robust in general, and we have had fewer deaths from pneumonia.

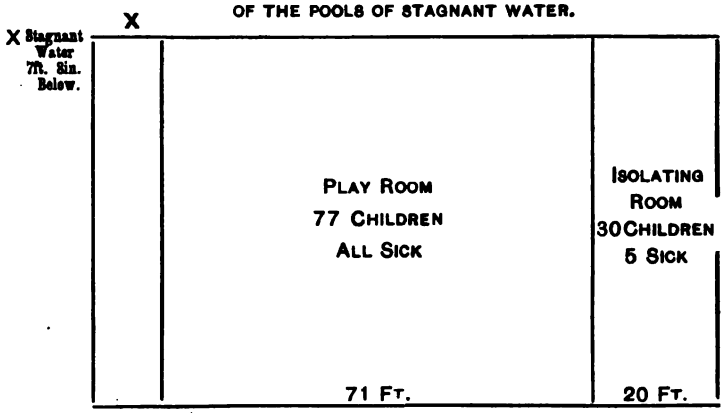
AN ACCOUNT OF A MILD EPIDEMIC OF UNCERTAIN NATURE IN CHILDREN.

BY ROWLAND G. FREEMAN, M.D.,

Clinical Lecturer on Pediatrics and Chief of Pediatric Clinic in the University and Bellevue Hospital Medical College; Attending Physician to the Foundling Hospital and the Seaside Hospital of St. John's Guild, New York.

In the Foundling Hospital, at the end of June, 1899, a group of seventy-seven children, who had been separated from the others by day, all became sick in a fairly similar manner at about the same time. These children were what are known as the "play-room children," children that during the day were taken to a large room on the ground floor of the building and there instructed in kindergarten work, and allowed to play under

PLAN SHOWING THE ROOMS IN WHICH THE SICK CHILDREN WERE, AND THE LOCATION OF THE POOLS OF STAGNANT WATER.



supervision. Adjoining this play-room, but cut off from it by a partition, is another smaller room used for isolating children with ring-worm of the scalp. In this room there were thirty children, five of whom became sick in a similar manner to those in the play-room, but less severely, so that none of them were transferred to the hospital. Of the seven hundred children in the Foundling Hospital at that time, none of the others became

sick in a similar manner, although on the floor above these two rooms there is a nursery which was occupied by children.

It became suddenly noticeable that the children in the play-room lacked their usual activity; they became drowsy, lost their appetites, and some of them complained of headache. These children became gradually worse; some of them would sleep sitting upright, others would lie down on the benches and sleep.

On June 28th two of these children had fever and seemed sick enough to be transferred to the hospital building and confined

Table of the Principal Symptoms and Physical Signs of the Hospital Cases

DATE	NAME	CHILL	HEAD-ACHE	VOMIT-ING	FACE FLUSH'D	TONGUE COATED	CONSTI-PATION	TENDERNESS OVER SPLEEN	BLOOD
June 28.	Mary A.			yes		yes		yes	neg.
" "	Joseph P.					yes	yes	yes	
" 29.	John Mc. C.	yes	yes			yes			plasm-odium
" "	James G.					yes	yes		neg.
" 30	Henry C.		yes	yes	yes	yes			neg.
" "	Aloysius I.		yes		yes	yes			
" "	John L.		yes	yes		yes			
" "	Anthony P.		yes			yes			neg.
July 1.	May M.		yes	yes	yes	yes			neg.
" 3.	Mary O'C.	yes	yes	yes		yes			
" 5.	George F.	slight	yes		yes	yes			neg.
" 10.	Mary L.	slight			yes	yes			

to bed. The following day, June 29th, two more, and on June 30th four more were so transferred, and one each on July 1st, 3rd, 5th and 10th.

Of the twelve children that were transferred to the hospital two had marked chills and two chilly sensation, eight complained of headache, one of abdominal pain and five vomited. They all had fever (see temperature charts) ranging from 100.5° to 105° and going up and down, some cases touching normal once each day, and others not. The pulse was accelerated, ranging from

100 to 140; the respirations in most cases were below 30, in some as high as 50.

On examination five had flushing of the face; they all showed coated tongues without the redness of the tongue and pharynx ordinarily associated with a gastroenteric disorder. One had abdominal tenderness. None had evident enlargement of the spleen, although two had tenderness localized over the spleen.

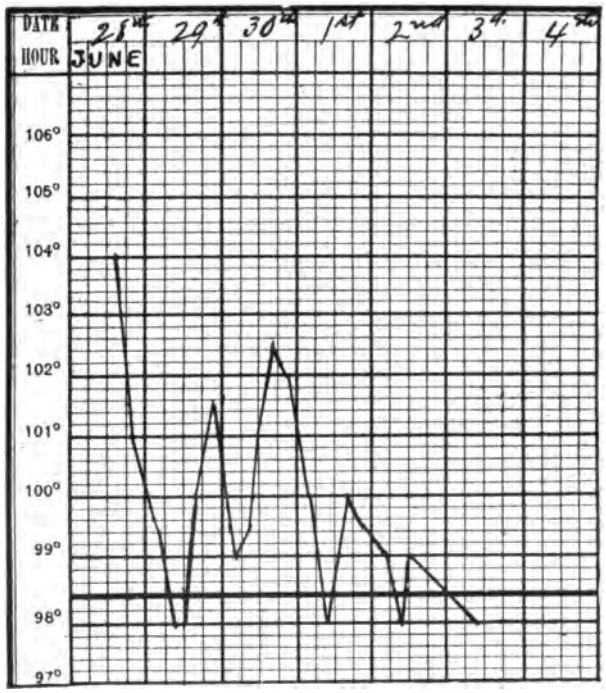


CHART I.—**Mary A.**

When the first 4 cases had been transferred, all coming from the same room and without the ordinary appearance and symptoms of acute gastrointestinal disorder, they were ordered castor-oil and were kept in bed on milk-diet for further observation.

The 5 cases that came into the hospital on the 30th, were of the same type. None of them had a history of bad bowels,

nor any evidence of gastroenteritic disorder, except that some of them had vomited and they all had the coated tongues.

After the initial purgative dose, with the rest in bed and milk-diet, some of the children showed a moderate improvement, but in none of them was there a subsidence of the fever or a recovery.

Examination of the blood by the house staff in seven of these children soon after admission gave a negative result.

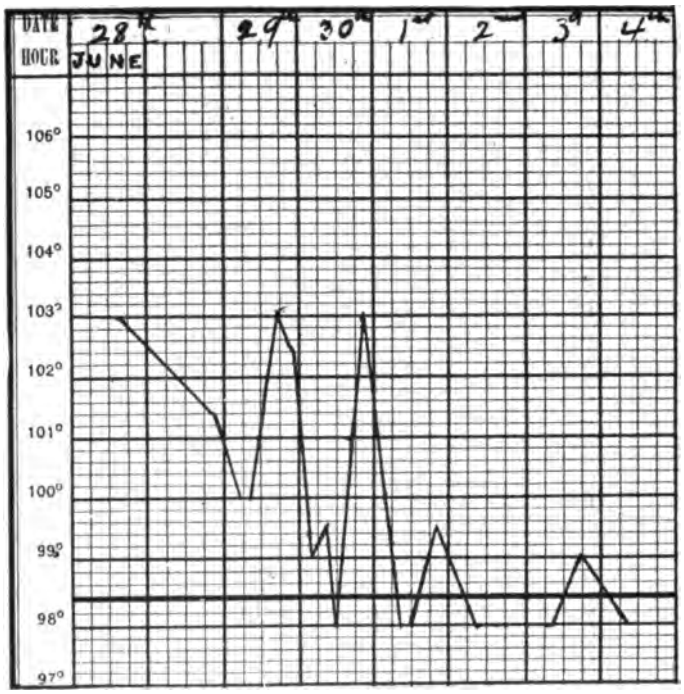


CHART II.—Joseph P.

On the first of July one of the children, John McC. (Chart III.), who had a chill on entrance, with a rise of temperature to 105°, followed by complete subsidence, had, on the third day, another rise to the same point, preceded by a chill. On making a second examination of the child's blood, the plasmodium was found. This typical manifestation, with the finding of the plasmodium in this case, seemed to render probable the suspected diagnosis of malaria in the other children, and immediately all of

those under observation in the hospital were given two grains of quinin every three or four hours, with a complete subsidence of the temperature in most cases within about twenty-four hours.

By this time the play-room children were becoming so much worse that their ordinary duties could not be fulfilled, and in my absence these children were all given quinin without first having obtained smears from the blood. They were at first treated with two grains of quinin a day, without amelioration, but upon the dose being increased to six grains a day, a rapid recovery of all took place.

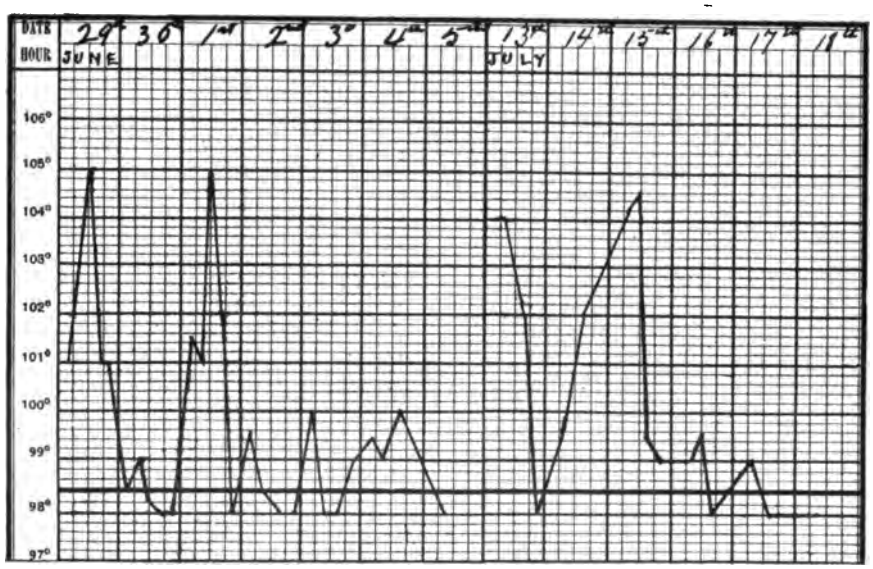


CHART III.—John McC.

The diagnosis of the condition present in these children is of considerable interest and that most readily reached in all the cases, except the one in which the plasmodium was found, is that of gastroenteric disorder. In support of this diagnosis we have the coated tongues, the headache, the vomiting in some cases and constipation in 2 cases; but against this diagnosis we have the fact that the children did not have red tongues or pharynges, that they did not have tympanitis or dilated stomachs, and not one of them had diarrhea, and the further fact that none of them cleared up entirely after acting on the bowels and

putting them on proper diet with rest. Furthermore, these play-room children had previously received the same food as the other children in the house, none of whom were affected. If then, there is doubt as to the existence of a simple gastro-enteric disturbance in these children, we are led to consider the other diagnosis of malaria.

The marked susceptibility of children to malarial infection has long been known but has recently been especially empha-

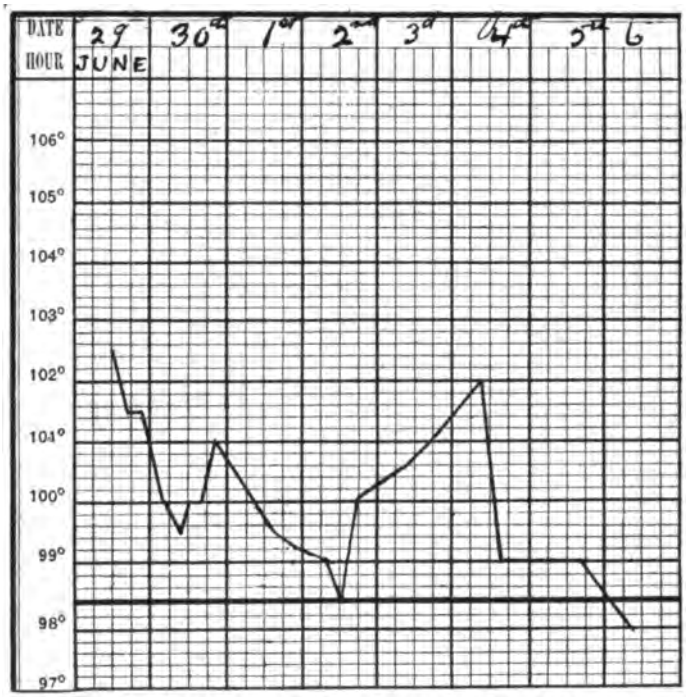


CHART IV.—**James G.**

sized by the work of Celli, Koch, and of the English Malaria Expedition to Sierre Leone. This susceptibility of children is so marked that Koch was in the habit of determining the extent of malaria in each new place he visited during his investigations by ascertaining the frequency of the malarial parasite in the blood of young children in the locality. This test he found to be accurate and reliable.

In those localities in which he found the blood of the chil-

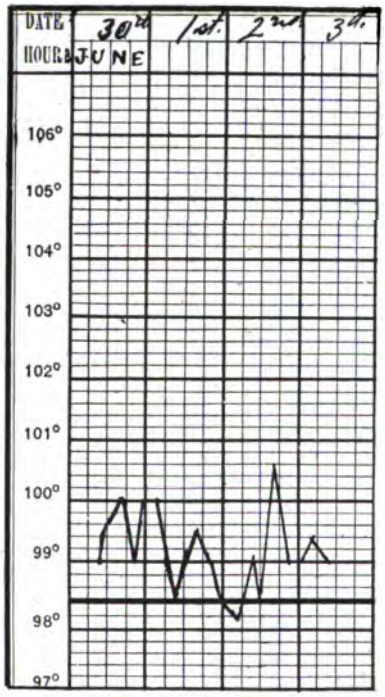


CHART V.—**Henry C.**

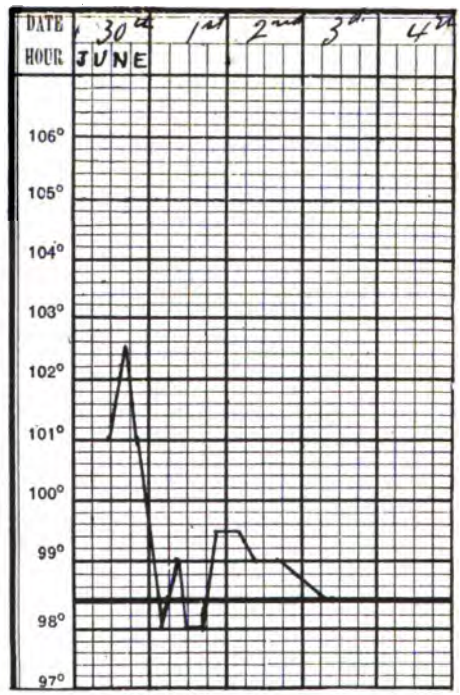


CHART VI.—**Aloysius I.**

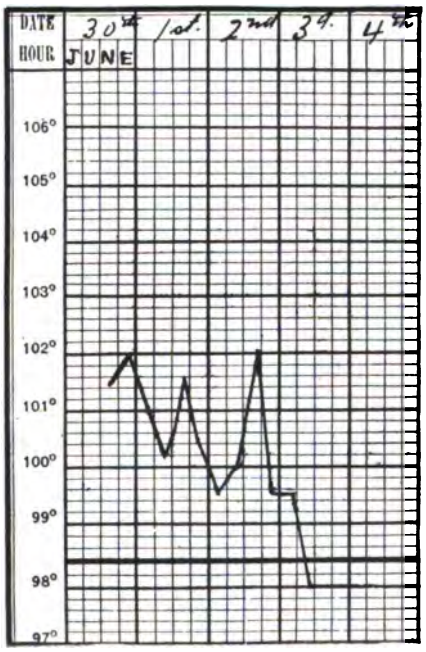


CHART VII.—**John L.**

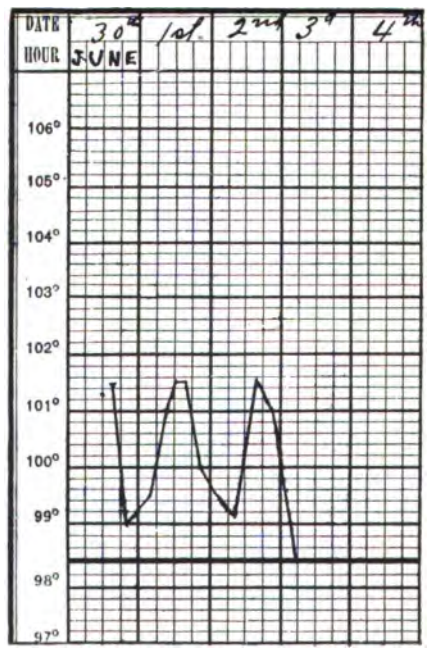


CHART VIII.—**Anthony P.**

dren ordinarily free from the malarial parasite, he found that no malaria existed in any of the population, while in the malarial regions he always found the young children very generally infected. This malarial infection was most common in children under one year of age, becoming less frequent with advancing years. Thus in one malarial district in which eighty-six children were examined for malarial parasites 16 per cent. of those under one year of age were found to be infected and 4 per cent. of those over one year of age.

In another village where one hundred and forty-one chil-

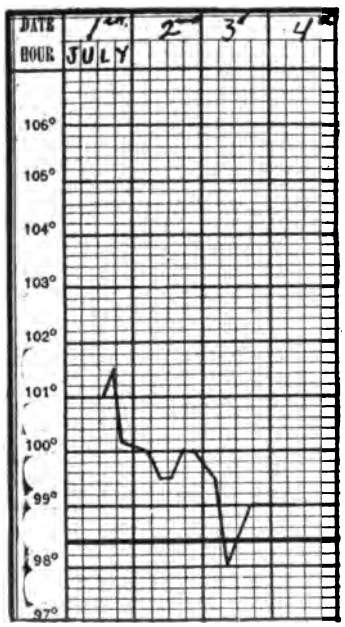


CHART IX.—**May M.**

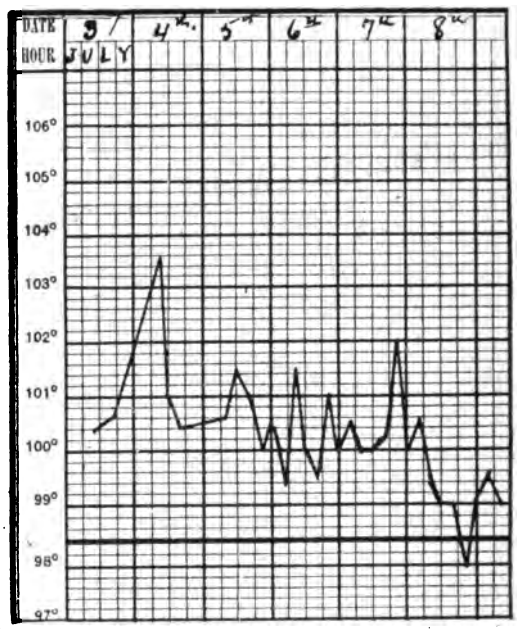


CHART X.—**Mary O'C.**

dren were examined 15.5 per cent. of those under one year of age showed the malarial organism in their blood and 7 per cent. of those over one year of age.

In still another village, of one hundred and eighty-one children examined, 41 per cent. of those under one year of age showed the malarial organism in their blood and 14.7 per cent. of those over one year of age.

It seems fair to conclude from such results that most chil-

dren are very susceptible to malarial infection and that immunity is gradually acquired, so that in this way the practical immunity of native adults in malarial districts may be explained.

The function of the mosquito as a carrier of malarial infection, and the importance of stagnant water as a breeding-place for the anopheles are so well established that they may be

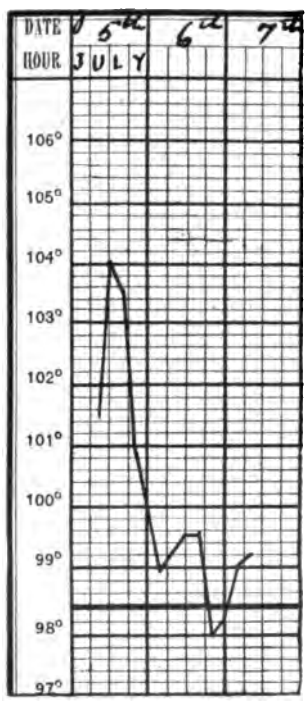


CHART XI.—**George.**

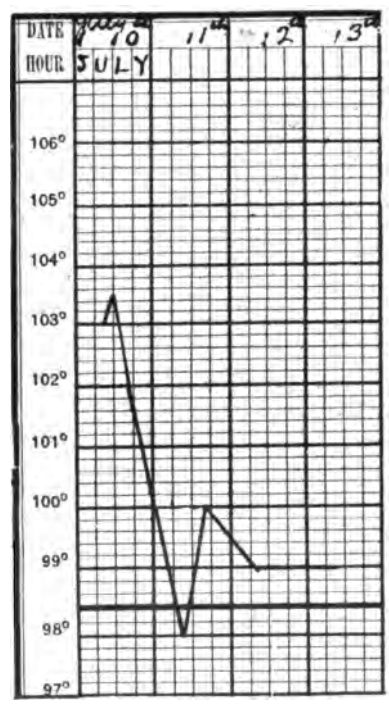


CHART XII.—**Mary L.**

merely mentioned as probably essential etiological factors in the production of malaria.

Clinically the condition of these children gave rise to a suspicion of malaria when first seen, simply by exclusion; they were unlike gastrointestinal disorder, they resembled each other closely but did not show the positive signs of enlarged spleen or of plasmodium, except in one case later. The temperature charts, with one exception, were not typical of malaria.

The question arises whether there is any stage in malarial infection in which the conditions found in these children may be present.

In studying the charts of cases of experimental malaria it is found that during the period of incubation, which lasts from one and a third to eleven days, there may be an irregular temperature without enlargement of spleen and without the presence of the plasmodium, this being followed, if curative treatment is not used, by the invasion with enlargement of the spleen, presence of plasmodium, and the typical temperature.

Assuming the diagnosis of malaria, we must search for the source of infection. It happened that in the spring of 1899 an excavation was made almost adjoining the play-room (see plan), which was nearly eight feet deep, preparatory to the erection of a new building. There had been but little rainfall during the two months preceding this epidemic. During the middle of May there had been about 7-10 of an inch rainfall; at the end of May 1-16 of an inch, and beginning with the 20th of June, on which day 1-10 of an inch of rain fell, there were a number of rainy days. On the 23d, 24th and 25th of June ½ of an inch of rain fell, while on the 28th and 29th 8-10 of an inch fell. During these rainfalls puddles of stagnant water collected in the bottom of this excavation. As will be noted this collection of stagnant water was nearest to the play-room and somewhat farther removed from the isolation-room. A reconciliation of this condition with the mosquito theory of the causation of malaria is, however, difficult, for the larval stage of the mosquito lasts from twenty to twenty-two days in summer, while there must still follow a period of eight to ten days after the mosquito has acquired malaria before these organisms may be transmitted to a person bitten.

It is further noteworthy that the temperature of the air during the early rains in May was not sufficiently high to facilitate the development of the mosquito larvae, and the rains at the end of June were too near to the time of the outbreak to allow for the development of the mosquito, and afterward of the malarial parasite within it. Moreover, no invasion of mosquitoes and no mosquito bites on the children were noted.

In conclusion I would say that these children answered clinically to a malarial condition in its prodromic stage more

nearly than to anything else, and that this diagnosis was further confirmed by one typical case of malarial seizure, accompanied by the presence of the plasmodium in the blood, and that all these eighty-two children recovered promptly on the administration of quinin; that as an etiological factor we find pools of stagnant water adjacent to the room in which these children were during the day; but when we attempt to ascribe this outbreak to these pools in the light of our present knowledge concerning the causation of malaria by mosquitoes, we find that the conditions present were not sufficient.

DISCUSSION.

DR. NORTHRUP.—In connection with the mosquito theory of malaria a colleague of mine, recently informed me that he had seen a case of estivo-autumnal malaria in a child eight months old, living in the most hygienic part of New York, in which mosquitoes played a part. He said that he asked the parents whether there were any mosquitoes about and they said, no, that the child was watched so carefully, a nurse being with it all the time, that it would be impossible for mosquitoes to approach it. While a blood expert was engaged making an examination he killed a mosquito on the child's forehead. Perhaps it is unfortunate that an autopsy was not held on that mosquito or at least that it was not captured.

DR. PACKARD.—I think the anophele appears in Philadelphia generally about June 15th; I don't know how early you may see it in New York.

It occurs to me that this epidemic may have been one of influenza and that the one case in which the plasmodium was found was a relapsing case of malaria. I have lately seen two such cases in Philadelphia where the malaria manifested itself during a period of lessened resistance brought about by influenza.

DR. KERLEY.—It has been my privilege during the past eight or ten years to do a great deal of out-patient work and I have frequently met with an affection with a history somewhat as Dr. Freeman has given. The child presents a coated tongue, a rise in temperature at a certain time each day; in some there is no fever, but a periodicity as regards the other symptoms. The child becomes languid and drowsy at a definite time every day,

usually in the afternoon. The examination of the blood fails to show the plasmodium and yet quinin cures the condition. I have not put these cases down as malaria and I do not know what they are.

It has been suggested that if we insert a needle into the spleen we might find the plasmodium.

DR. BUCKINGHAM.—It seems absurd for a Boston man to say anything about malaria before men who see so much more of it at home, but during the Spanish War I had charge of a number of men who had malaria and it was not at all uncommon to make a blood examination with negative result, and yet later to find plasmodia abundantly in the same case. Those cases were all of the estivo-autumnal type and repeated examinations would at first be negative.

DR. WENTWORTH.—I would like to ask Dr. Freeman how old some of these patients were and particularly the age of the one that had the plasmodium.

DR. FREEMAN.—All were between three and five years of age.

DR. WENTWORTH.—I notice that the one with the plasmodium is the only one that had a typical malaria temperature chart.

DR. ADAMS.—I do not see the evidence of malaria in these cases and I am accustomed to see a good many cases of malaria in very young children. The observation of Dr. Acker and myself is that we almost invariably find the plasmodium in children whenever there is a marked paroxysm, and when we get such a marked paroxysm it will be controlled by quinin at once. It is certainly a singular thing that but one of that large group of cases showed the plasmodium and it rather tends to vitiate the diagnosis of malaria in epidemic form.

Another point is that those temperature charts are not the typical temperatures as we usually see them; 101° to 102° rectal temperature does not represent a malarial temperature to my mind. If Dr. Freeman's charts showed a rectal temperature of 104° to 107° in children with paroxysms and a sudden drop in the temperatures without convulsions I should be more inclined to accept his malarial theory. It is difficult, too, to reconcile with the malarial theory the fact that these cases all occurred at the one particular time. That is not our experience with malaria either in hospital or private practice, and I should rather be inclined to the theory advanced by Dr. Freeman, but which he did not hold, that there must have been some gastro-enteritic disorder, that there must have been something that all the children took to give them this slight rise of temperature. A rectal

temperature of 101° is very slight for the child's normal rectal temperature is more frequently 100° than it is 99° and I would not consider these temperatures representative of any particular disease.

DR. FREEMAN.—I did not hold particularly that this was a malarial epidemic and the more I have thought of it the less sure I felt about it. The temperature given here, if they are malarial, are of the periods of incubation, except in 1 case. These patients were all cured by quinin and they were not relieved at all by the efforts to clean out the bowels and remove any possible toxic substances.

MULTIPLE ARTHRITIS IN A CHILD TWO YEARS OLD SUFFERING FROM GONORRHEAL VULVOVAGINITIS.

BY GEO. N. ACKER, M.D.,

Washington, D. C.

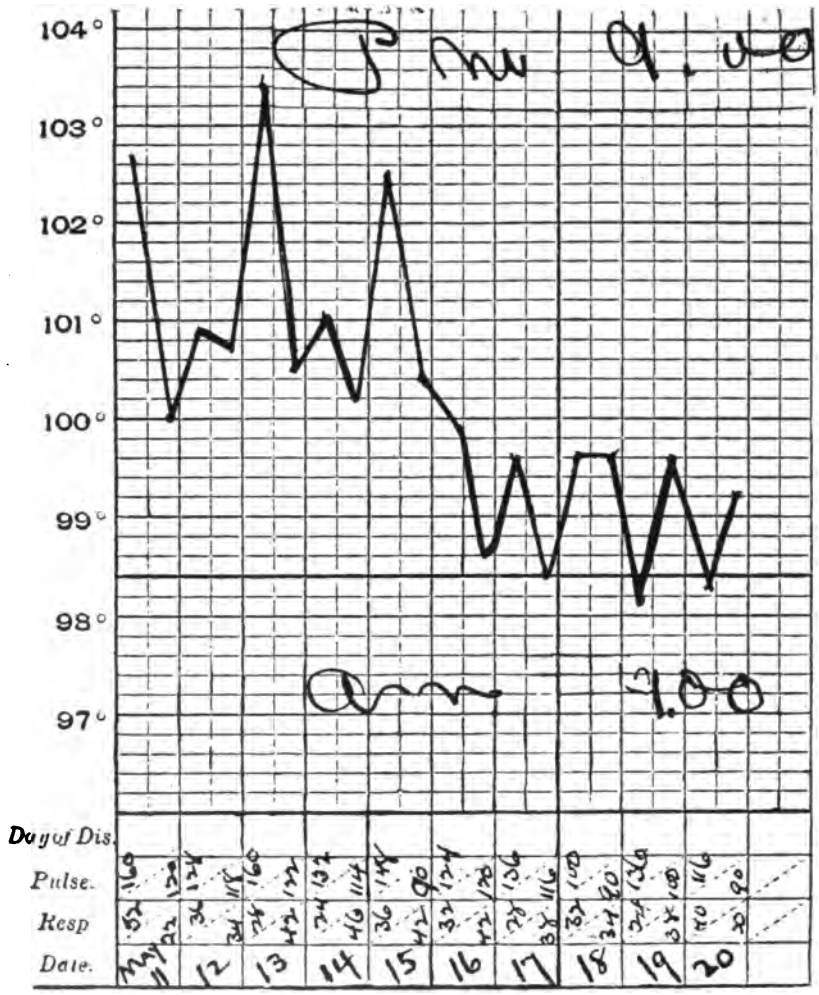
V. C., colored female, two years of age, came under the care of my assistant, Dr. Wm. N. Fisher, in the Children's Hospital Dispensary on May 1, 1901. She had a profuse vaginal discharge which was found to contain numerous gonococci. This discharge had attracted the mother's attention several days previously, but had probably existed for some days longer, for the child had complained of a pain when she urinated. At this visit when the lower limbs were handled they appeared to be painful but were not swollen. She returned to the dispensary in a few days with the discharge about the same and the ankles and knees markedly swollen and painful. Until this time the child was able to walk about. The case was referred to the hospital for treatment and she entered my service May 11th.

FAMILY HISTORY.—Father and mother are both living and in good health. No specific nor tubercular history.

PREVIOUS HISTORY.—Born after a difficult labor, forceps used. Breast-fed for fifteen months then put on cow's milk, tea, etc. Dentition began at eight months. Had always been a very healthy child, having had none of the infectious diseases of childhood.

PRESENT ILLNESS.—Mother not definite about the date when the child was taken sick, but she first noticed a profuse creamy vaginal discharge about two weeks ago, accompanied by pain-

ful and frequent micturition, with pains in back and loins. Seven days later the child began to have severe pains in the right ankle, the joint becoming red, hot and swollen. It was extremely sensitive to pressure and the movements were lim-



ited and painful. The next day the right knee became involved and after that the left knee and ankle. The condition has grown progressively worse. She has had fever. Bowels constipated, appetite good.

PRESENT CONDITION.—The child is well developed and unusually intelligent for her age. Both knees and ankles are swollen and painful, the patient crying whenever disturbed. The right knee shows the greatest amount of swelling, the joint being symmetrically and uniformly enlarged. The right ankle and left knee and ankle are involved to a lesser extent, motion being exceedingly limited and painful. Teeth and gums are in a good condition. Tongue coated. The examination of the heart, lungs, liver and spleen showed them to be in a normal condition. The child appears very weak and exhausted and complains of pains in head and limbs, does not sleep well and is cross and irritable when disturbed. There is a free purulent vaginal discharge. The external genitalia are swollen and there is an intense hyperemia of the vulvovaginal mucous surfaces. The urine contains a slight trace of albumin with many epithelial cells and leucocytes. No renal casts. The inguinal, cervical and axillary lymph nodes are slightly enlarged.

TREATMENT.—The girl was placed on liquid diet and 5 grs. of salicylate of soda given every four hours.

Vaginal douches of warm saturated solution of boric acid were ordered every three hours, the joints to be wrapped in cotton after the use of a 25 per cent. ichthyol ointment twice daily.

May 14th.—Slept better than on previous nights. There is a small amount of discharge from the vagina. Douches continued night and morning and in the interval a tampon of gauze saturated with a 1 to 4 ichthyol-glycerin solution was kept in the vagina.

The right knee somewhat swollen and the other joints are almost normal in size. To-day the right knee joint was aspirated and a small amount of serum was obtained, which on examination showed many large polynuclear leucocytes, but no gonococci.

May 17th.—Much improved. The salicylate of soda was stopped and 5 drops of mild tincture of iron given three times daily.

May 24th.—The girl is bright and uses her legs as if free of

pain. The right knee is still enlarged and though the joint is movable yet the leg cannot be fully extended.

The urine is negative. There are some shreds in the douche but no discharge.

June 7th.—No vaginal discharge. No signs of swelling about joint. Motion good and the girl is able to walk.

HARE-LIP.

BY B. K. RACHFORD, M.D.,

Cincinnati, O.

The purpose of this paper is to relate the strange and tragic history of a family into which there were born four girls with hare-lips and cleft palates, and three boys not showing any trace of these deformities.

Both Mr. and Mrs. A., the parents of these children, have family histories of tuberculosis. Mr. A.'s mother, one uncle and two aunts on his mother's side, died of this disease. Two of his brothers also died from tuberculosis, and of two remaining sisters both are probably tuberculous. Mr. A. himself and one brother show no evidence of this disease.

Mrs. A. lost one aunt, and possibly another, from tuberculosis, but Mrs. A., when I first knew her, was apparently in perfect health. There is no history of hare-lip or cleft palate in the family, but the mother has a high-arched palate.

This family came under my observation in 1893, when the mother was approaching her fifth confinement. At this time I obtained the following history.

In 1881 there was born to Mr. and Mrs. A. their first child, a girl, with a cleft through the upper lip and nostril, and hard and soft palates. This deformity was very great and could in no way be explained by them, either through heredity or maternal impressions. This child was operated upon when a few weeks old, obliterating in great part the deformity of the lip. She is now living, a young woman of twenty; the cleft in the palate is great, and the lip is still deformed enough to destroy the symmetry of the mouth.

In 1883 another girl baby was born to them, which also had a cleft through the lip, nostril and hard and soft palates. The deformity in this infant was greater than in her sister. She was operated upon shortly after her birth and is now living, a young girl of seventeen. The cleft in the palate amounts to

almost the loss of the whole roof of the mouth. The lip was partially restored, but the deformity is still very great.

In 1889, as the mother approached her third confinement she became confident that her baby was to be a boy and that he would be "all right." A little later a baby boy was born, and the mother's happiness was beyond control. He showed no evidence of hare-lip or cleft palate, and was perfectly formed in every way. This child, however, did not thrive and finally, after months of illness, died of tuberculosis when nine months old. The mother's grief at the loss of this child was very great, and many were the friends on all sides who advised her not to have any more children.

In 1891, however, she became pregnant for the fourth time, and during her pregnancy was in a state of great nervous excitement because of her fear that her fourth baby would be a girl, and that it would have the same deformity. Her fears were realized, a baby girl was born, and the deformity was so terrible that the child lived only a few hours, and was never seen by the mother. In this infant the father says that "half of the child's face was gone" and that "through this opening the pulsating base of the brain could be seen."

The mother came under my observation in 1893, when she was pregnant with her fifth child. It was at this time that I obtained the foregoing history.

I saw the mother at intervals during this pregnancy, and strange to say, she manifested no nervousness as to the result. She was sure her baby was to be a boy and that he would be "all right." I attended the mother in this confinement, and when a baby boy, free from all deformity, was born, it was difficult to restrain her in her wild delight. This baby thrived only fairly well for two years, and during this time the mother's devotion to the child and fear that something might happen to him was the chief drawback to his healthful development. The baby, during his waking and part of his sleeping hours, lived in her arms, and it was almost impossible to give him the fresh air that he needed, because she was in constant dread of his "catching cold." But notwithstanding these drawbacks the child continued in fair health until shortly after he was two years of age, when he died of tubercular meningitis.

It is impossible to describe the grief of the mother at this time. For many months she lost all interest in her household

affairs, and was thought by her friends to be losing her mind. The dead boy remained her idol; she thought and talked of nothing else.

In 1895 the husband on my advice took his wife to California. In 1896 she returned from there pregnant with her sixth child. At this time she had recovered her self-control and seemed again in good health. During this pregnancy she was haunted with the same nervous fears that preceded the birth of her last girl. She was afraid this baby would be a girl and that it would be deformed, as all her other girl babies had been. I attended her in this confinement, and sure enough, her worst fears were realized. A baby girl was born, with a double hare-lip, a floating intermaxillary bone, and a complete cleft of the palate. Immediately the child was born she discovered by my manner that the worst had happened, and she went into a violent hysterical paroxysm. For a number of days after the birth of this baby she did not care to see it. The deformity was such that the baby could not nurse. It, therefore, had to be fed artificially, and the mother, quite crazed with grief and disappointment, insisted that it should be kept in another room. After the fifth day, however, she begun to manifest some interest in the infant, and at the end of three weeks she was caring for it as tenderly as she had for any of the other children.

When this baby was three weeks old the intermaxillary bone was removed, the cleft palate and hare-lip operated upon, and a very good result was obtained, the deformity in great part being obliterated. This child continued to thrive until she was about eighteen months old, when she contracted tuberculosis by direct and constant exposure to this disease, and died four months later. The contagion in this instance was readily traced to a brother of Mr. A. who came to his house to die with tuberculosis, when this baby was about fourteen months old. The brother lived but a few months, and during all of this time the child was constantly exposed to the contagion, and commenced to manifest the first symptoms of tuberculosis within a few weeks after his death.

A few months later Mrs. A., who had nursed her brother-in-law and baby through tuberculosis, commenced to decline in health. For a time her husband thought her ill-health was due to grief. She continued, however, to fail in health, until she developed active symptoms of pulmonary tuberculosis.

When this occurred the family moved again to California. Here the mother lived an out-of-door life for about a year, and apparently recovered her health. All of the active symptoms of tuberculosis disappeared, and in 1900 she came home pregnant with her seventh child. I did not see her this time until a few days before the birth of her baby, at which time it was evident that she had not recovered her health completely, and was much frailer than she had been in her previous pregnancies. She was, however, in the happiest frame of mind, notwithstanding the tragedies with which her life had been filled. At this time again she was sure that she was to have a boy, and that all would be right. She received a few days before her confinement a letter from an aunt telling her how terrible was the responsibility she was taking in bringing children into the world, when the chances were that they would be deformed. But notwithstanding such influences as these, she remained sublimely confident of the result, and during her labor manifested not the least nervousness or anxiety. A perfect boy baby was born, and when she was thus informed she received the news with as little excitement as any other mother might. During this pregnancy at least she had such faith in her "presentiment," that when a boy was born she manifested only the natural pleasure of a mother at a time like this.

This boy is now two months old and is thriving fairly well, but the mother herself has developed an active pulmonary tuberculosis and is now preparing to leave for California for the third time.

The above family history is not only extremely interesting, and terribly tragic, but I think it is also quite unique in some of its features, and presents some problems in heredity which it is difficult to solve. And while it is not my intention to attempt this solution, yet I wish to call attention with emphasis to the following facts:

There is no family history of hare-lip or cleft palate, but the mother has a high-arched palate. There is a very bad family history of tuberculosis.

The four girls all had cleft palates and hare-lips.

The three boys were entirely free from this deformity.

The first and second boy died of tuberculosis, and the third, an infant now two months old, has not as yet lived long enough to contract this disease.

Only one of the girls died of tuberculosis, and in that instance the contagion was so direct and so great that it would have been almost impossible for any predisposed child to escape.

There was at no time any claim on the part of the mother that maternal impressions had played any role in producing these deformities. The deformity of her first-born was a great surprise and sorrow to her and caused her to look forward with fear and dread to the birth of her second child, which was also deformed; but notwithstanding this experience she expected with much confidence that her third child would be a boy and not deformed, and this expectation was realized, but the birth of the fourth child, which was deformed, was looked forward to with dread.

Of her last three pregnancies I can speak from personal observation and have no hesitancy in saying that during the fifth and seventh pregnancies Mrs. A., was quite confident that boys would be born and that they would be without deformities, and this confidence gave her composure, but during her sixth labor she was nervous and had to exercise considerable self-control to keep herself from becoming hysterical over the fear that a deformed girl baby would be born to her.

The mother herself does not believe that her self-control and confidence in the result had anything to do with the sex or deformities of her children. On the contrary, she believes that in some intuitive way she was informed during her third, fifth and seventh pregnancies that her babies would be boys and that they would be "all right." This intuitive knowledge grew with her experience and was the cause of her composure during these pregnancies, but it had nothing to do with influencing the result.

In connection with this history the following quotations will be of interest because of their bearing on the etiology of hare-lip.

In Guy's Hospital Reports, 1861, it is stated that "hare-lip occurs more commonly in boys. Forty-seven cases are reported, 30 boys and 17 girls; of these same cases 21 were simple hare-lip, 10 boys and 11 girls; 5 of the cases had fissures of the hard palate, 3 boys and 2 girls; 17 of these cases had hare-lip and a complete fissure of both palates, 13 boys and 4 girls. There were also 4 cases of double hare-lip, all in boys.

It will be noted that in these cases the more severe the deformity the greater the preponderance of boys."

In St. Thomas' Hospital Reports, 1876, it is stated that in most cases there is no history of heredity. Yet a few exceptions are given. Two sisters, one with a simple cleft and the other with a double hare-lip, had a grand-aunt with a cleft palate. In another instance a father and child had cleft palates. In this family there were three normal children. In another family the father and two children had cleft palates, and a third child had hare-lip.

Samuel D. Gross* says "hare-lip is sometimes associated with other congenital mal-formations. I have witnessed its coexistence with club-foot, bifid-spine, scrotal hernia and deformities of the hands and fingers." Concerning the sex Gross says, "I have seen it as often in one sex as in the other." He also quotes a case communicated to him by Dr. Lightfoot, of Kentucky, in which this malformation has shown itself in four successive generations.

Francis Mason† in a comprehensive review of the subject of hare-lip says: "There has been much speculation as to the causes of hare-lip. As a rule it is attributed to some shock or impression made upon the mother during pregnancy. Sir Wm. Ferguson, with many others, puts no faith in such views, and adds that he has often detected a partial defect in the upper lip and jaw of one or both of the parents, and since he directed my attention to this point, I have observed it in many instances.

"I am myself somewhat slow to believe in the theory of maternal impressions, but I may allude to a case in which the coincidence was so great as to leave little doubt that such impressions may have some influence in producing the deformity.

"In November, 1874, a child with single hare-lip was brought to me at St. Thomas' Hospital. The mother stated that while she was riding in an omnibus she was shocked at the appearance of a boy who had had hare-lip and who had been operated on in early life. She had only been married a month, and spoke to her husband, mother and sister of her fear that if she had a baby it might have hare-lip. When the child was born her first inquiry was, 'Is he all right, or has he got a hare-lip?'"

Again, in one of Dewar's cases of hare-lip the mother of

* System of Surgery.

† St. Thomas' Hospital Reports, 1875.

the child resided previous to her marriage near a boy who had been operated on for hare-lip, and she was perfectly familiar with the child's appearance. Soon after she became pregnant with her second child she was strangely impressed with the idea that her infant was to be born deformed.

"Hare-lip seems in many instances to be hereditary, for it is not very uncommon for a surgeon to operate on two or three members of a family. Liston operated on four, and in a case of mine a mother and child were both successfully operated on for hare-lip.

"Demarguay related a case at the Surgical Society of Paris, in which, from the grandparents downward, eleven children have been born with hare-lip. The same surgeon refers to the case of an infant affected with double hare-lip, whose mother had the same deformity. This woman had seven children, four of whom had hare-lip.

"A correspondent in the *British Medical Journal* also states that in his family hare-lip has been handed down from one branch of the family to another for the last hundred years. Dr. Bellingham, of Dublin, operated upon two brothers, whose relations on both sides had been similarly affected, and these were all males; thus their father's grandfather had hare-lip, and a second cousin of their father's was similarly affected. On the mother's side two of her second cousins, both boys, had hare-lip."

William Rose in his excellent monograph, "On Hare-lip and Cleft Palate*" says:

"As to the etiology of these defects, but little is known. Heredity is an undoubted factor in their production, and an investigation of the family history will in many cases elicit a confirmation of such an idea. Thus in two instances in my own practice I have been able to determine that the father, grandmother and great-grandfather had all suffered from hare-lip to a greater or less extent.

"An examination of the parents' mouths should always be made, and very commonly it will be found that one or both possess a short upper lip and a high arched narrow palate. In others there is a slight groove in the alveolar process between the central and lateral incisors. I have also observed a small symmetrical crease on either side of the medium line in the

* London, 1891.

upper lip, indicating a tendency to, if not a natural intrauterine cure of, a double hare-lip.

"The so-called maternal impression is looked on, especially by the laity, as another common cause of these deformities. Medical men will usually receive histories of such with a smile of incredulity, and rightly so; but some recorded cases are so definite that to condemn such an explanation too dogmatically seems scarcely to indicate a scientific spirit.

"Manley reports a case of a child born with hare-lip. The mother of this child, when four months pregnant, was startled by a boy running into her arms, from whose face blood was streaming. She saw a cut in the left side of the upper lip extending through its substance into the nostril, laying bare gums and teeth. She turned faint with fright, and could not banish the thought even after reaching home.

"The union of the parts entering into the formation of the palate, alveolus and lip is normally completed by the eighth to the tenth week, and when once this has occurred, no maternal impression (such as seeing a gashed lip) could, as far as we know, bring about a retrogressive change. Should some shock occur to the mother prior to that period, we can fully appreciate the possibility of its interfering with a typical growth of the parts then being produced. That a severe shock to an infant may produce coincidentally a lamellar cataract and defective development of dentine is well recognized; that a similar type of shock acting on the mother should result in defective union of parts, developing at that period in the fœtus, is not strange, but that the real shock and the so-called maternal impression are one and the same, is more than doubtful."

A CONTRIBUTION TO THE SYMPTOMATOLOGY OF CRETINISM AND OTHER FORMS OF IDIOCY.

BY HENRY KOPLIK, M.D.,

Attending Pediatrist to the Mount Sinai Hospital,

AND JACOB LICHTENSTEIN, M.D.,

New York.

Stigmata of degeneration are defined as deviations from the normal physical conformation of the individual, which are seen for the most part in degenerates; that is in individuals morally and mentally deficient. If these stigmata are studied more fully we find that as a rule they rarely affect the organs necessary to the maintenance of life. They are found in those portions of the body least necessary to the continuance of life. These stigmata are of a nature found in an early embryonal period of man or even in a lower order of the animal world. Many of these so-called stigmata are seen so frequently in persons in other respects normally developed, that we can scarcely regard them as signs of degeneracy. Others, however, are truly characteristic, if not pathognomonic.

In the latter class we would include an anatomical sign or stigma which we have for some time carefully studied and which we have observed on certain patients of our clinical material. While demonstrating the peculiarities of the skin and conformation of the hand of cretins, we were struck with a prominence in the region of the antithenar eminence and over the situation of the os pisiformis. Inasmuch as this peculiar eminence was present in all the cretins which we have met we at first thought that this was a peculiarity of the cretin hand. On further study, however, we became convinced that this sign or stigma was to be found in other degenerate individuals. Before

pointing out the cases in which this peculiar stigma is found we will describe it more fully.

This prominence is immediately adjacent to the groove which separates the palm of the hand from the forearm. It is distinctly localized to this part of the antithenar eminence and, viewed from the side, rises abruptly from the above groove, giving a bayonet-like appearance. This prominence is apparently an over-development or hypertrophy of the small muscles

Fig. 1. CAST OF THE HAND OF A BOY CRETIN FOUR YEARS OF AGE UNDER TREATMENT FOR TWO YEARS AND SIX MONTHS. *E* EMINENCE DESCRIBED IN THE PAPER.

of the inner border of the hand, attached to the os pisiformis, as well as perhaps an enlarged condition of this bone itself. A similar prominence can be seen on the forefoot of the domestic cat. Inasmuch as this animal supports the weight of the body in walking on this prominence, we might conclude that in these children walking as they do late in life, this prominence is the result of a hypertrophy caused by the act of crawling on the floor and supporting the weight on the inner border of the hands.

But this prominence has been observed by us in a cretin only three months of age.

We are therefore justified in assuming that this prominence over the pisiform bone in degenerate infants and children is a congenital anomaly.

It would be of great interest to know whether this prominence exists in the hand of the upper extremities of the highly developed anthropoid apes and whether it is more highly de-

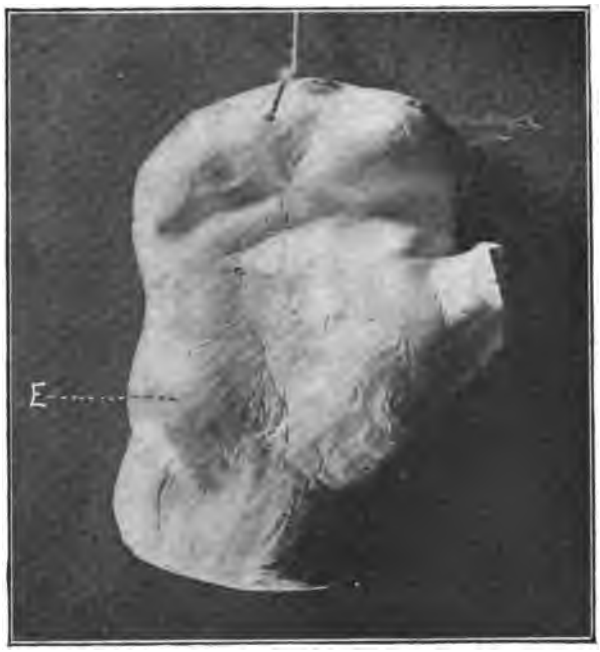


Fig. II. CAST OF THE HAND OF A FEMALE DWARF TWELVE YEARS OF AGE, WITH ENLARGED THYROID GLAND AND MENTAL WEAKNESS. *E* EMINENCE DESCRIBED IN THE PAPER.

veloped in those species of apes who walk on all four extremities. As for the occurrence of this prominence which we have described, we have not as yet seen it in a perfectly normal infant or child. The infants in which we have seen this prominence were idiots, cretins, microcephalic idiots, and children who were congenitally deficient. We have as yet not met this anatomical prominence in the Mongolian idiot, though our experience includes a large number of cases of this affection. We

found it in all cases of cretinism. (See Fig. I.) It was absent in a case in which there were certain physical signs seen in cretins, but in which the child was quite intelligent. It was seen in two children who had marked macroglossia with polydactylia. Both these children were markedly intelligent.

This anatomical stigma of degeneration was found in 2 cases of microcephalic idiocy. It was also found in an idiot of hydrocephalic class, and in a dwarf of marked mental degeneracy with goitre. (See Fig. II.) The cases have not been numerous enough to allow of any further conclusions. We are convinced, however, that the prominence just described is a true anatomical stigma of degeneration, and as far as the cretinism is concerned a highly interesting part of the symptomatology of this disease.

A CASE OF MEASLES COMPLICATED BY APPENDICITIS.

BY HAROLD WILLIAMS, M.D.,

Boston.

M. P., aged twelve years, first complained of feeling ill on March 31st. He was never a strong boy and had frequently suffered from attacks of gastrointestinal disturbance. He had had measles some years previous. The symptoms of present attack consisted of nausea and general malaise. Temperature 100°. He was put to bed and dieted.

April 1st.—He was less comfortable though no new symptoms appeared. Temperature 101°.

April 2nd.—Patient complained of a slight sore throat and vomited once. Temperature 102°.

April 3rd.—I first saw him. Patient not so comfortable as upon previous days. Temperature 103.4°. Had vomited once or twice; complained of abdominal pain; cough, weak eyes and sore throat. Inspection: conjunctiva slightly injected; mouth red with striking congestion of alveolar processes; congested appearance of posterior pharynx and a catarrhal inflammation of right tonsil to which uvula was adherent. Careful examination of the abdomen showed some tenderness in different places; always fleeting and never localized.

April 4th.—Well-marked eruption of measles on face and behind ears. Eruption confluent; conjunctivitis more marked; cough still slight; abdominal symptoms as before; temperature 104.4°.

April 5th.—Early in the morning patient complained of pain in right side. This pain steadily increased in severity. At 10 A.M., when I saw him, it was exactly located at a point an inch above Poupart's ligament; it was increased on the slightest pressure. The locality was indicated by patient with one finger. There was neither dullness nor induration. The diagnosis of an acute inflammation of the appendix was made; Dr. Arthur T.

Cabot was called and the case was placed in his charge. It was also thought advisable that Dr. J. H. McCollom should give us the benefit of his wide experience in measles; a disease, the association of which with a fulminating appendicitis in a boy of delicate physique seemed a matter of some gravity. Dr. Cabot, in view of the probable rapidly advancing process, decided upon immediate operation in which decision Dr. McCollom and I concurred. The operation was performed by Dr. Cabot two hours later with the assistance of Dr. Hugh Cabot.

April 6th.—Temperature fell to 99° during night and remained at this point throughout a remarkably satisfactory convalescence.

Pathological examination of the appendix by Prof. Mallory is as follows:

"April 6, 1901.—Appendix. Gross examination. Appendix 4.5 cm. long, varied from .6-.8 cm. in diameter. Somewhat swollen in middle two thirds. Serous coat injected, especially at tip; fresh fibrin adherent in places, forming irregular yellow areas. Mesentary injected. Split open nearly to tip; contains a fecal concretion the size of a small bean. Proximal end of mucosa swollen, lumen quite small and partly filled with creamy tenacious purulent material; beyond this is a definite enlargement of the lumen in which the concretion closely fits; the mucosa here is dark red to greenish, and is evidently becoming gangrenous; beyond this dilatation, the mucosa is thickened and there is some muco-purulent secretion.

"Anatomical Diagnosis. *Acute Appendicitis.*

"Microscopic examination of a longitudinal section shows an acute exudative process most marked on the outer surface of the appendix. The mucosa is fairly well-preserved, but is infiltrated in places with numerous polynuclear leucocytes."

(signed) F. B. MALLORY.

The event proves, so far as we can judge from a single case, that the complicating presence of measles is not a contraindication for the operation of appendicitis under circumstances in which the highest surgical skill is obtainable. The combination of the two pathological processes is, so far as I know, a rare one.

PULMONARY GANGRENE IN AN INFANT.

BY WALTER LESTER CARR, M.D.,

New York.

A. J., one year old, was admitted to the Infants' Hospital, Randall's Island, on October 18, 1899. Dr. Appleton, of the House Staff, obtained the following history:

The baby was born on October 12, 1898, and abandoned by the mother when six months old. The father was a white man, the mother a colored woman. During the summer of 1899 the child had bronchitis and "bowel trouble"; the exact character of the latter could not be determined. There was no history of any other disease. Dentition began when ten months old. For about three months there had been a great deal of perspiration of the head during sleep.

Examination on admission showed the infant to be fairly nourished. The skin was clear but of a more sallow color than was accounted for by the race. The fontanelles were quite wide open, the head square; epiphyses enlarged; slight beading of the ribs and lateral curvature of the spine. There were only two teeth, the two lower incisors. The heart was normal; the left lung was normal; the right lung showed poor expansion and slight dulness in the lower lobe behind, but no râles were present. The liver was slightly enlarged; the spleen was not palpable; the temperature was normal.

October 19th, 7 A.M.—Temperature 103.4°F. As the baby was constipated, the colon was washed out with a high enema; the result was a yellow stool with a great quantity of mucus. The temperature fell until at 1 P.M. it was 100°; at 5 P.M 99.2°. While pulse and respiration seemed unmodified, no trouble was detected with the lungs.

October 20th to 28th.—Child well during the whole time. Temperature normal, appetite good and food well digested. On the 28th there was a greenish yellow stool and a slight rise of temperature. Physical examination negative.

October 30th.—Temperature 101°; bowels rather loose, with

mucus in the stools; pulse and respiration normal. There was a slight cough. The colon was irrigated.

October 31st.—Temperature about the same, varying from 100° to 102°, highest at 4 A.M. Pulse strong, 128 to 136. Respiration remained at 28. Stools continued to show mucus. The temperature dropped under colon irrigation. The child was fretful. No change in the lungs.

November 1st.—Temperature 100° to 103.5°, highest at 7 A.M.; pulse 128 to 136; respiration 28 to 32. Stools about the same, apparently no change by irrigation and a dose of castor oil. Some abdominal distention. Cough rather more frequent, with vomiting of mucus. Child very restless. Physical examination showed exaggerated breathing over the whole of the right lung. No râles detected, no dulness. Left lung, mucous râles.

November 2d.—Temperature 100° to 100.2°; pulse 132 to 134; respiration 34 to 36. Stools green and yellow, free of mucus. No abdominal distention. Cough about as before noted. Baby took food well, no vomiting; slept quietly. Physical signs the same.

November 3d.—Temperature ranged from 100° to 103°, the temperature being 103° at 2 A.M., 100° at 4 A.M. and 103° at 6 A.M.; respiration 32 to 36; cough still frequent. Vomited mucus but retained food. Baby was very restless all day. A few mucous râles over right lung in front and posteriorly; vocal fremitus increased over the right lung, with dulness at the base and slight dulness at apex. Bronchial breathing at the right base and bronchovesicular at the apex. Left lung was normal except for a few soft râles at base.

November 4th.—Temperature 99.2° to 102°; pulse 130 to 132; respiration 28 to 34. Baby appeared better. Physical signs about the same.

November 6th.—Temperature ranged from 99° at 6 A.M. to 103° at 11 P.M.; pulse and respiration not increased over the previous days. The baby moved head constantly and moaned. The stools were normal but there was some vomiting. Pulse not of good quality, so that strychnin and whiskey were given according to indications.

November 7th.—Temperature 101° to 103.6°; respirations 28 to 36; pulse not above 128 and stronger. Physical signs showed in front diminished expansion over the right lung, with higher pitched expiration and a few mucous râles. Behind over

the right lung, vocal fremitus increased, dulness extended from spine of the scapula to the base of the lung. Intensified voice sound, expiration prolonged and a few fine crepitations heard at the base. In the left lung hyper-resonance noted in spots below angle of scapula. Areas of dulness with fine râles; occasional mucous râles.

November 8th.—The baby dull and apathetic; took about two ounces of food at a feeding; retained it during the morning but vomited it at night. General physical condition as recorded.

November 9th.—Temperature from 100° to 102.4°; the latter at 3 A.M.; pulse and respiration about as noted at other times; very restless; cough more frequent; respiration shallow. Physical signs the same except that the voice sounds were less exaggerated and crepitant sounds were heard in posterior axillary line, and also anterior to the area over which they were formerly noted.

November 10th.—Temperature from 102° to 105.2° in the morning 105° at 9 P.M.; respiration and pulse about the same as on other days. Respiration was not so labored. Physical signs were the same except that the crepitation was more marked over the posterior surface of the right lung. The stools contained mucus and undigested casein, and were somewhat offensive.

November 11th.—Baby was dull; extremities cold; vomited once in the morning; stools offensive; pulse was irregular.

November 13th.—Cough was easier; child expectorated considerable mucus without odor. In the early morning was very restless; later in the day dull and weak. Vomiting seemed to be occasioned by the cough.

November 15th.—No vomiting; abdomen much distended; respiration labored; pulse weak; head slightly retracted; right leg drawn up; reaction to stimuli very slow; there did not seem to be complete analgesia nor anesthesia. Physical signs as noted.

November 16th.—Quiet but weaker; slept a great deal; retained nearly all the food; cough was frequent; pulse rapid and weak; respiration not labored; some strabismus and pupils reacted very slowly. Fontanelles depressed; head slightly retracted; the right leg drawn up. Physical signs in the lungs seemed about the same. No resolution was to be detected.

November 17th.—Less evidence of cerebral irritation.

November 18th.—Infant more apathetic; cough not trouble-

some; respirations from 30 to 40, not so shallow; pulse weak. Irritative symptoms slightly increased; more depression of the fontanelles, retraction of the head and strabismus. The right leg flexed on the abdomen, but no paralysis. Physical signs: the left lung showed more consolidation at the base and the right lung at the apex.

November 19th.—Temperature 100° to 104°; pulse 120 to 138; respirations 28 to 40. Patient very weak and dull; took very little food; stools very dark and loose, but without any undigested material. Abdomen much distended, not relieved by irrigation; coughed very little, could not expectorate; respiration labored; pulse weak; increased depression of fontanelles; strabismus, reaction to light very slow; head retracted. Baby lay most of the time in a stupor. Both legs were flexed on the abdomen. Reaction in left leg normal, in right very slow; no paralysis, no convulsions. Death at 8 P.M.

At no time during the child's illness was there any fetid odor of the breath, neither was the expectoration fetid. The sputa were white or whitish yellow, had no green nor brown tinge and were not streaked with blood. The baby, however, raised much more than was usual for one of its age. The character of the temperature curve and also the general condition pointed to a septic process, but this could not be definitely determined to be a gangrene in addition to a septic bronchopneumonia. It was thought that the symptoms were increased by the disturbed digestion and the intestinal catarrh, which persisted almost the whole period of the pneumonia.

AUTOPSY REPORT.—The autopsy was performed about eighteen hours after death.

BODY of a poorly nourished mulatto child, without skin lesions.

BRAIN.—Pia very cloudy, especially over the cerebral cortex; blood vessels moderately congested; ventricles normal.

HEART.—Normal.

LUNGS.—No pleurisy; *left lower lobe* almost completely solid with red bronchopneumonia, and containing, in its posterior half, a darker, brownish, softened (gangrenous) area 2½ x 1 x 1 inches in diameter, extending to within one-eighth inch of the pleural surface. Several small pulmonary veins in this lobe contained soft, dark thrombi; *upper lobe* shows a small area of gangrene just above the posterior, inferior border, but it is

in an earlier stage than that in the lower lobe. Edema throughout both lungs was very marked. *Right lower lobe,* acute bronchopneumonia occupying about half the lobe, with a gangrenous area about half as large as that in the left lung; *upper lobe,* bronchopneumonia in the apex only. *Bronchial lymph nodes* small and red.

LIVER.—Marked degree of fatty infiltration.

SPLEEN.—Normal.

STOMACH.—Post-mortem softening of entire cardiac end.

INTESTINES.—Increased amount of mucus throughout; no ulcers; in colon the solitary follicles were swollen. Mesenteric lymph nodes normal.

KIDNEYS.—Gray, capsules free, cortex not thickened, markings fairly distinct.

ANATOMICAL DIAGNOSIS.—Bronchopneumonia; pulmonary gangrene; fatty liver; catarrhal entero-colitis.

The literature of pulmonary gangrene is quite exhaustive, but most of it dates from the time of Rilliet and Barthez (*Traité des Maladies des Enfants,* 1843), who were the first to study the subject with care. They had 11 personal cases and were able to find 5 in literature, reported by Constant, Chavigner and Berton, the references, however, being omitted. In the same year Bondet published (*Arch. Gen. de Med.,* 1843, Vol. II., p. 385), 5 other cases, making the material amount to 21 at that period. In the edition of Rilliet and Barthez's work dated 1884, reference is made to a thesis by L. Atkins (*Ueber Gangrena Pulmonum bei Kindern,* Zurich, 1872), and this is doubtless the author quoted by Eustace Smith, as having reported 31 cases.* The authors speak of 26 cases in making calculations, this number probably representing their personal experience.

Vogel and Biedert report a case in a boy of fourteen, where the gangrene had lasted six years after pneumonia due to the pressure of a head of wheat in a bronchus. The case was ultimately cured by operation.

West's case (*Diseases of Children*) was that of a three-year-old girl in poor general condition, who developed pneumonia and died after an illness of two weeks' duration. At the autopsy the bronchopneumonia was found to be limited especially to the

* In a personal communication, Eustace Smith states that he obtained his information from the inaugural thesis of Atkins. It seems impossible to get a printed copy.

right lung, and a gangrenous cavity was present in the upper lobe.

McNalty (*Med. Times and Gazette*, 1872, Vol. II.,) describes a case of pulmonary gangrene with pneumonia following measles in a boy three-and-a-half years old. There was extreme fetor of the breath and expectoration, which contained clotted blood and debris from the lungs. On the day preceding death he vomited blood and also passed clots by the bowels. The autopsy showed pneumonia and gangrene in both lobes of the left lung.

Von Muralt's case (*Corresp. Blatt. für Schweiz. Aertze*, 1882, No. 10) followed empyema, the child being only two years old. Recovery resulted after incision. An older patient was observed by Day (*Practitioner*, 1885), a girl of ten, in whom fetid breath was a marked symptom during an attack of pleuropneumonia which ended fatally. The autopsy disclosed two abscesses in the right lung, containing fetid pus and surrounded by gangrenous lung tissue. There were areas of pneumonia and gangrene throughout the entire lung. Henoch's case followed typhoid fever in a child of four years; hemoptysis was present. Ewart and Benham (*Lancet*, 1887, Vol. I.,) also report a case following typhoid fever and occurring with empyema. The boy recovered, perflation having been used and fetid pus and shreds of gangrenous lung tissue removed.

The case which Holt observed (ARCHIVES OF PEDIATRICS, 1885) occurred in a boy three-and-a-half years old who presented the symptoms of pleuropneumonia. At the autopsy gangrenous areas were found in the right lung. There had never been any fetor of the breath.

Pasteur describes (*British Medical Journal*, 1888, Vol. II.) the illness of a seven-year-old boy whose pulmonary gangrene was probably due to the passage of a foreign body from the esophagus into the bronchus, an opening between the two having been found at autopsy. Fetid breath and hemoptysis were prominent symptoms, and a cavity of the right lung was diagnosed. An incision was made in the right second intercostal space, one inch from the sternum. A large quantity of putrid fluid and gangrenous lung were expelled. Improvement was marked at first, but death occurred thirteen days after operation; it had probably been done too late—in the fifth week of the illness.

De Jersey's case (*Lancet*, 1892, Vol. I.) followed pneumonia and empyema in a child twenty-one months old. The breath

and the discharge from the empyema wound were very fetid. At autopsy, gangrenous areas were found throughout the entire left lung, but no pulmonary embolism.

Thomas (*Rev. Méd. de la Suisse Romande*, 1895, Vol. XV.) reports a case which recovered after an attack of pneumonia and pulmonary gangrene evidenced by fetor of the breath and sputum; the latter was very abundant. Empyema developed and the pus had an offensive odor. Incision resulted in cure.

A case of diffuse, latent pulmonary gangrene has been reported by A. Martinez Vargas (*The Jacobi Festschrift*, 1900). The patient was a three-year-old child with pneumonia; hemoptysis occurred, but the breath was not fetid. The author lays great stress upon the fact that the marked general symptoms were out of all proportion to the pulmonary signs. The gangrene was found at autopsy.

Seitz (*Kurzgef. Lehrb. d. Kinderheilk.*, Berlin, 1901) found pulmonary gangrene almost as common in children as in adults. It may follow any of the acute infectious or respiratory diseases, infectious emboli or foreign bodies in the lungs, or chronic intestinal catarrh. As for the bacteriology, any one of the pyogenic group of bacteria and some saprophytes may be found in the gangrenous areas.

Seitz says that the most striking diagnostic point, the gangrenous odor, does not differ essentially from that which accompanies bronchiectasis and putrid bronchitis. It is apparent from the case reported in this paper and from the literature that the gangrenous odor is by no means a constant symptom of pulmonary gangrene in young children, even when the necrotic process is quite extensive, as in Vargas' case.

A positive diagnosis of the condition is of importance in view of the treatment. Herczel (*Wien. Med. Presse*, 1900) collected 91 cases of pulmonary gangrene which had been operated upon, with 61 per cent. of recoveries. He believes that the expectant treatment, which almost invariably leads to a fatal ending, is no longer justifiable. Gangrene following croupous pneumonia gave the best results by operation, and that due to perforation of the esophagus the worst.

PULMONARY GANGRENE.

BY FRANCIS HUBER, M.D.,

New York.

During the past two years 3 fatal cases of gangrene of the lung were met with in the children's service at the Vanderbilt Clinic. In one, a boy of seven, the physical signs lead to the conclusion that both sides were implicated. Death finally occurred. No autopsy was permitted.

In the second, a child of some three years of age, seen at irregular intervals, there was a large cavity on the right side, and though the breath was extremely fetid, the amount of trouble on the other side was considered to be of such a grave nature that operation was not thought advisable. The case terminated fatally.

In the third case, a child of four years of age, considerable improvement took place under the use of guaiacol and tonics. The family were opposed to operation and subsequently we learned that the child had died rather suddenly and unexpectedly. No autopsy. Unfortunately the notes in these cases are imperfect, as we could not keep the patients under close observation.

In a fourth case the termination was more favorable. A boy of some five years of age was presented with a history of having been squeezed in a gate while eating some peanut candy. As his symptoms developed about this time the mother suspected that some of the candy had accidentally entered the larynx.

There were marked evidences of localized trouble in one lung and the odor of the breath was somewhat offensive; general condition fair. Under tonic treatment the patient improved. Some three weeks later the grandmother brought the boy to the children's clinic with the report that a day or so before he had coughed up a portion of a peanut. From this time on,

there was a rapid disappearance of the pulmonary signs, with complete restoration of health.

In the following case, though it could not be definitely verified, a small gangrenous focus with abscess was suspected. For a number of months an undersized girl, five years of age, with large adenoids was kept under careful observation. In the beginning a pleuropneumonia of the right lower lobe was diagnosed. The high leucocytosis and the physical signs, five weeks after the onset, with but slight displacement of the apex to the left, lead to the belief that a localized purulent collection had taken place. Exploratory puncture was performed upon three occasions at intervals of a few days. The first attempt revealed a bloody fluid, the second some turbid sanguineous liquid and the third was also negative.

For some time a peculiar, sweetish, offensive odor was noticed in the breath, sufficiently marked to be appreciated several feet from the patient. It gradually subsided and finally could only be noticed after a severe paroxysm of coughing. It may be stated that the cough was usually spasmodic in character and particularly severe at night—at no time was there a whoop nor was whooping-cough present in the other members of the family.

Under careful nursing, tonics of iron, guaiacol, creasote, etc., the unpleasant symptoms subsided and a fair condition of health was established. Though some pleural thickening persists, it is not possible to detect any dilated bronchus or cavity.

Localized gangrenous processes in the lungs, unsuspected during life and unattended by fetid breath and other symptoms characteristic of the condition, are now and then found on autopsy. Vargas, in the Festschrift in honor of A. Jacobi, reports a striking instance under the title "Gangrena Pulmonar Difusa Latente," in which the characteristic sputum and odor to the breath were absent. A correct diagnosis was not possible until the autopsy revealed the true condition.

Some cases of empyema may be attributed to this cause. A number of years ago such a case terminating favorably came under personal observation. The true nature was not suspected until a large piece of necrotic pulmonary tissue was washed away in irrigating the pleural cavity—such being the routine practice at that time.

Finally another case may be briefly referred to, though

the cause of the trouble was not evident. Operative interference at a time when the outlook was not very favorable, was followed by the happiest results.

Benny S., three years old, born in the United States of Russian-Polish parents, has always lived in the thickly populated section of the eastern district, a veritable ghetto in New York City.*

THE FAMILY HISTORY is unimportant, nor has the early history of the patient any particular bearing upon the present trouble. For a number of months prior to his admission to the "Jacobi Ward," he had been troubled with cough at times paroxysmal in character and frequently attended by vomiting—no whoop or other symptoms pointing to pertussis. Temperature was of an irregular type, chilliness and sweating reported. Loss of strength and flesh considerable. A short time before admission, the attending physician, suspecting pus, made an exploratory puncture with negative results. Soon after the mother claimed to have observed pus in the sputum and a decidedly offensive odor to the breath. As he did not improve at home, he was advised to be sent to the hospital and was admitted May 14, 1900, pale, emaciated, hectic in looks, weighing about twenty-three pounds. Pulse weak and rapid, temperature 102°, respiration 52. Cough severe and paroxysmal, strong fetid odor to breath, considerable degree of dyspnea. Outlook not very promising.

PHYSICAL EXAMINATION.—Right lung, posteriorly, dulness above over upper lobe and in axilla to about seventh rib; coarse respiration and bronchophony over dull area. Anteriorly, dulness generally. Breathing somewhat exaggerated over apex; fine rales about the level of third rib; dry rales in axilla. Left lung, posteriorly, fine and moist rales (small and large); bronchophony present, particularly marked over lower lobe. Anteriorly, over second rib, bronchial breathing with coarse rales. In axilla respiratory murmur feeble, otherwise unchanged. Hemoglobin 50 per cent.—r. c. 5,000,000. w. c. 19,600. Sputum, elastic fibres, otherwise negative; no bacilli.

TREATMENT was supporting and symptomatic. In about a week the right lung had cleared up pretty well and the general

* Those familiar with Manhattan Island will readily recognize the crowded and unsanitary condition prevailing here, though they are greatly improved compared to what they were ten years ago.

condition was more favorable. The physical signs varied and though a local abscess with gangrene was suspected, its position could not be exactly made out. June 9th after a careful, physical examination, an exploratory puncture posteriorly on the left side below the angle of the scapula revealed thick pus a short distance below the surface. It is to be regretted that a bacteriological examination was omitted.

Two days later under chloroform narcosis, Dr. Hubbard, the house physician, removed a section of the ninth rib and exposed the pleura. Exploratory punctures in various directions failed to reveal the site of the abscess. The general condition of the child not being very good, it was concluded to tampon the wound and await developments. In cleaning up and dressing the child the discovery was made that a quantity of pus had escaped through the nose and mouth, the abscess cavity having emptied itself during the time of operation.

Subsequently within the next few days several punctures were made and though no pus was found, it was noticed that each insertion of the needle was followed by the escape of a small quantity of air, offensive in character. No pneumothorax present at any time. A small drainage tube was therefore inserted. Next day some fetid pus discharged and the amount gradually increased. On the 23d a portion of the adjoining rib was resected to facilitate drainage and a second tube inserted. From this time on, the condition gradually improved. Because of the offensive odor from the wound, similar to that emanating from the mouth, the cavities were irrigated. It was observed that when any force was employed, a paroxysmal cough would be induced and the fluid would come up through the mouth. Improvement was gradual and about July 6th the boy began to walk about ward. From this time on the improvement was rapid. The odor became less and less offensive, the discharge diminished and the rubber tubes were replaced by drains of horse hair. August 29th the upper sinus was healed and on the 31st the lower. Patient was discharged well on September 5th with good expansion. General condition excellent.

In the early part of the year he was again presented. There had been no return of his trouble. His health was good and with the exception of the scars of his wounds, there were no physical signs of the serious condition through which he had passed.

Suppuration of the lung with gangrene cannot be regarded as a disease *per se*; the symptom complex ought rather to be looked upon as a complication occurring in a variety of states in which necrotic areas undergo putrefactive changes.

It is not easy to explain the comparative infrequency of the condition, for germs of different degrees of virulence are always present in the air passages. As a rule the lower lobe is more frequently affected and the lesion is peripheral rather than central.

A lowered tissue resistance, the result of local or general causes is frequently noticed. In the case of Benny S. there was no bone disease or cardiac lesion present, that might have given rise to pulmonary embolus, nor could any history of typhoid be elicited. Aspiration pneumonia, particularly when due to foreign bodies of animal or vegetable origin, rapidly takes on putrefactive changes and causes local gangrene. Diabetes and bronchiectasis could be excluded. In the absence of any of the recognized conditions, the gangrene in this case must be attributed to the debility resulting from preceding protracted pulmonary changes of an obscure nature.

MONSTER PER DEFECTUM.

BY A. C. COTTON, M.D.,
Chicago, Ill.

Through the courtesy of Dr. J. M. Lang I am able to present to you this specimen which he delivered. He gives the following history. "I examined Mrs. R., aged twenty-four years, American, primipara, supposed to be pregnant five months and found no signs of fetal life. Twenty-four hours later pains began terminating in the delivery of a dead fetus. (No complications). The mother has always enjoyed good health

Fig. 1.—GENERAL APPEARANCE OF THE MONSTER SHOWING DEFECTIVE EXTREMITIES AND THE WIDE SEPARATION OF THE PARIETAL BONES.

with the exception of the ordinary diseases of childhood. Her parents are still living and well. She has two sisters who have borne normal children. Her husband is thirty years old, Hebrew, saloon-keeper and a steady drinker. He is apparently in good health and gives no history of syphilis or any disease other than those of childhood. His parents and brothers are living and well.

"Two months after delivery, an examination of Mrs. R.'s urine showed it to be smoky in color, with albumin, hyaline

casts and epithelium. Under treatment she made rapid improvement."

The specimen was kept in 96 per cent. alcohol for two months before it came into my possession. The resultant hardening and shrinking made measurements and weights of little value.

It will be seen at a glance that we have here an abnormality of development—a monster by defect. The most marked defect is the shortness of the extremities, especially of the lower. (Fig. I.) At first view it would appear that the arms and thighs are wanting, the forearms and legs arising from the shoulders and hips respectively.

Fig. II.—SHOWING PECULIAR WEBBING AND WIDTH OF THE HAND.

The skiagram, however, shows but one bone in each extremity, probably the humeri and femora, each presenting a marked curvature. No traces of forearm or leg bones are to be found.

The hands and feet show peculiarities in their breadth, the brevity of their digits, a queer clubbing of their distal extremities and a partial webbing, (Fig. II.) The position of the little finger —particularly of the left hand—is almost at a right angle to its neighbors, and the great toe, especially the left, stands apart from the others, appearing more like a thumb.

The wide separation of the parietals, the sulcus extending deep between the frontal bones, with the large size of the head,

suggests a previous hydrocephalus. The disappearance of the fluid and subsequent collapse of soft tissues caused the deep groove seen in the specimen. (Fig. I.) Upon opening the cranium, a cursory inspection shows the cortical substance attenuated as by pressure, and collapsed ventricles, further suggestive of hydrocephalus. The remnant of a blood clot underneath the right parietal is noted.

A very noticeable anomaly is seen at the umbilicus, (Figs. I., and III.), in the form of an omphalocele, with the funis at its

Fig. III.—TONGUE-LIKE PROJECTION OF THE LIVER AND OMPHALOCELE.

upper border. This hernia is the size of a plum, its outer layer being continuous with the amniotic covering of the cord. Section reveals the contents of this sac to be a coil of intestine and a tongue-like projection of the right lobe of the liver. (Fig. III.) The peritoneal surface of the sac and its contents are smooth and free from adhesions. The large size of the liver, even for a fetus, is noticeable.

Upon opening the thorax the first incision penetrated the pericardial sac, fine pleural adhesions being extensive. Similar adhesions, but firmer, were found over the thymus. The thy-

mus is large and very irregular in shape, terminating above in a tit-like projection which overlaps the thyroid isthmus.

The integument, thick and covered with lanugo, presents a dense leathery consistency, with a deposit of fat, rarely found in a fetus of five months.

As to classification, this monster, like many others, presents a combination of features peculiar to itself. The shortened limbs would class it as a phocomelus. The absence of certain bones of the extremities would place it in the sixth division of Hirst's adaptation of Saint Hiliare's classification, viz.: anomaly by numerical defect. The omphalocele brings it into the class celosoma.

The absence of these bones of the extremities, or of their ossific centres and the failure of union of the ventral plates would locate the etiologic disturbance early in the second month of embryonal life, or before. It is probable the hydrocephalus developed later, as its earlier existence would have resulted in a monster of an anencephalic type.

Certain features, as the broad squat appearance, bluntness of outline, thickened, brawny integument, excess of subcutaneous connective tissue and the heavy prolabia suggest myxedema, but this has no obvious relation to the class phocomelia.

While at the first glance the shortened extremities with apparently normal length of trunk call to mind achondroplasia, still the osseous deficiencies both in extremities and cranium would rule this out.

A CASE OF VERY PERSISTENT LARYNGEAL STENOSIS.

BY J. P. CROZER GRIFFITH, M.D.,

Clinical Professor of the Diseases of Children, in the University of Pennsylvania, Philadelphia.

The following case of laryngeal stenosis, or what may, perhaps, better be called laryngeal stridor, seems sufficiently unusual, and of a nature obscure enough to warrant placing it upon record:

Lily S., age twenty months, white, began about February 15th to suffer from difficulty in respiration. She was attended by the family physician, who, after some time, pronounced the disease laryngeal diphtheria and sent her to the Municipal Hospital on March 12, 1901. Here, according to information received, she had no exudate visible in the throat or larynx, the nares showed no evidence of diphtheria, and two cultures on the second and the ninth day of her stay were negative. Her voice was husky, almost inaudible, and the respiration was slightly embarrassed. Her laryngeal symptoms improved, but had not entirely disappeared at her discharge after twelve days. Then respiration gradually grew worse again, and she was brought to the Children's Hospital about seven weeks after the beginning of the illness.

April 5, 1901.—On admission she was suffering from severe dyspnea, which was always present although varying in intensity. With respiration at its worst the child was decidedly cyanosed and had an anxious expression of face. At its best she would sit up and handle toys, yet even then there was free playing of all the accessory muscles of respiration.

April 6th the following note was made: Breathing slightly better although still very labored with both inspiration and expiration. There is extreme retraction of the epigastrium, together with sinking in of the sternum and bulging of the cartilages, attending each inspiration. The child's head is extremely rachitic, but there is no beading of the ribs or en-

largement of the wrists. The color is fairly good. Chvostek's facial symptom is absent.

Dr. Walter J. Freeman, laryngologist to the hospital, made a laryngoscopic examination on this date and reported that the cords were reddened and swollen, and that the condition appeared to be one of subacute laryngitis with thickening. There was no membrane visible, and no reason to suspect papilloma or paralysis.

Up to the 9th of April the dyspnea continued unabated in spite of the constant employment of vapor and the administration of strong sedatives. Upon the 9th the condition was so threatening that intubation was performed by Dr. J. H. Jopson. This gave immediate relief; yet the distortion of the chest by the dyspnea had been so marked that it was not until the next day that the depression of the sternum disappeared.

The patient wore the tube from the 9th to the 19th of the month, except for the coughing of it up on a number of occasions, after each of which dyspnea and cyanosis always returned, necessitating prompt replacing of it. On the 19th the tube was removed during two hours and then replaced. On the 27th it was removed permanently. On the 29th the breathing was only slightly affected and the child was sent home.

May 4th, the child was seen in the dispensary. She had been doing well, having stridor only when coughing or swallowing.

May 5th, the child was entered again in the wards, suffering from pneumonia. From this she recovered, and during the attack there was little affection of the larynx.

May 20th, there was only a very husky voice present, but no stridor. Chvostek's and Trousseau's symptoms were absent.

The previous family and personal history add to the interest of the case. Repeated questionings did not shake the mother's statement that about August or September, 1900, the child suffered in a similar way for about four weeks, the stridor being persistent although not nearly so severe as in this last attack. Further, that she is one of several children in the family, and that five others suffered at times during infancy, "when cutting teeth," from symptoms of troublesome stenosis, lasting for perhaps a couple of weeks at a time. The affection in all of them was different from ordinary croup in that it

persisted uninterruptedly both night and day. The sound of the cough was often like the noise made by a "rooster."

The interest of this case centres, of course, in the diagnosis. Pressure on the trachea or bronchi by large tuberculous bronchial lymph nodes might readily have caused persistent stridor. So, also, probably, could pressure by an enlarged thymus gland; but in neither case could intubation have given complete relief, followed, on each occasion that the tube was coughed up, by return of the dyspnea. This fact places the stenosis pretty clearly in the larynx. Pressure of enlarged lymph nodes upon a branch of the pneumogastric nerve has been assigned as a cause of reflex laryngeal spasm, or, in some cases, of laryngeal paralysis; but as Marfan points out, it is difficult to see why a unilateral pressure such as this is likely to be, should cause a bilateral disease. Moreover the occurrence of two attacks in the child's history and the recovery from each excludes this condition.

Of all the affections of the larynx the one most prominently suggesting itself when the child was admitted to the hospital was, of course, diphtheria. But the absence of any bacterial growth in the cultures, the fact that the laryngeal examination in neither hospital showed any membrane and, more than all, the history of the attack excluded the disease.

An abductor paralysis after a precedent diphtherial was equally impossible for the same reasons. Papilloma of the larynx would scarcely have produced dyspnea so severe and so persistent as this case exhibited, unless the mass had been so large that the laryngeal examination would have readily exhibited it. In such an event, also, such marked improvement could scarcely have taken place. Edema of the larynx can hardly be seriously considered in the absence of any disease producing it, and in view of the fact that examination of the larynx showed nothing of the sort discoverable. Laryngismus stridulus, in the ordinary sense of the term, can be readily excluded. The chief diagnostic characteristic of this disease, upon which writers seem to agree with great uniformity, is the occurrence of brief, severe laryngeal spasms with intervals more or less long of complete absence of laryngeal symptoms. This was completely absent in the case now reported.

Finally we have to consider acute laryngitis with spasmodic symptoms, such as is exemplified in many cases of false croup.

As well known, there often lasts in this disease for a day or two a slight tendency to persistent stridulous respiration. Why this should be present in some cases and absent in others seems to depend upon the innate tendency of the individual child. We have also the well-known cases of acute laryngitis consecutive to measles, in which there is persistent, severe stenosis of the larynx, accompanied by fever, often requiring intubation, and not infrequently fatal. In the case under consideration we certainly had present on the first examination an intense congestion of the mucous membrane of the larynx. It seems very probable that in this case there was also a specially marked personal and family tendency to laryngeal spasm, such as is often seen in many families in which croup occurs. The family history accounts in part for the neurosis. Still more does the remarkable development of rachitis as shown by the shape of the child's head, although the other bony evidences of it were not marked.

In fine, then, we probably have to do in this case with an acute or subacute laryngitis with which was associated a remarkable personal and family tendency to laryngeal spasm, allied to laryngismus stridulus, and depending in part, like it, upon rickets, but differing in its tendency to persistence.

The diagnosis is, however, only provisional and open to question.

DISCUSSION.

DR. ROTCH.—I think these cases belong to a very important class that should be investigated, and should be reported as often as possible. They are difficult to diagnose, and on that depends, of course, the treatment. I would like to suggest that besides the conditions described, that of congenital stridor should be considered. Cases have been reported, and are quite authentic, where in babies you have almost a choreic condition of the glottis and upper part of the pharynx. They last for some time, occasionally for months. Congenital stridor occurs only in the first few months of life and the peculiar sound is made continuously. In the cases described by Dr. Griffith we at times suspect a laryngitis of the Klebs-Loeffler variety, and yet, when cultures are made as far down the throat

as you can get, the result is negative. Laryngoscopic examination even by an expert may result in the report that nothing can be seen. I have seen one such case in a baby six or seven months old where the dyspnea was extreme, where the cultures were negative and where nothing was seen in the larynx and no other clinical symptoms of diphtheria existed, and yet the autopsies showed diphtheria. Perhaps all of us have met with young babies where diphtheria existed deep in the larynx without the usual clinical evidences. As we must recognize the difficulty of making a diagnosis in these cases, I wish to insist that antitoxin should be administered.

A case almost identical with Dr. Griffith's came under my care with this difficult breathing without any apparent cause. It appeared to be a mild laryngitis in an excessively neurotic baby. It was treated with a continuous atmosphere of steam, and it was not necessary to perform intubation or tracheotomy. It was a simple case of laryngitis. Of course, those cases are unusual. In these cases you are met with a difficult problem, for you do not wish to operate unless it is absolutely necessary, and in such a case where the diagnosis is necessarily difficult, even if the cultures are negative, you should not be afraid to use antitoxin.

DR. BUCKINGHAM.—I wish to report a case that came into the Children's Hospital six weeks ago: a colored child, admitted for a very marked condition of rickets. It developed this laryngeal condition, and an examination of the secretion showed nothing but staphylococcus. It was given antitoxin with no benefit for a time, but after ten days it improved, and I suppose it was a case of ordinary laryngitis with a great deal of edema of the larynx. I do not think it was a diphtheria case because it had a good pulse, which is unusual in diphtheria.

DR. ROTCH.—I think you may get absolutely negative symptoms at times in babies with laryngeal diphtheria, and even the pulse is not a good indication.

DR. KOPLIK.—In some of these cases of laryngismus stridulus the spasm is not altogether confined to the larynx. I understood Dr. Griffith to say that after the intubation tube was introduced in his case the epigastric retraction did not disappear until the next day, and this makes me think there was possibly not only a spasm of the larynx but a spasm of the diaphragm.

DR. GRIFFITH.—The question of the use of antitoxin did not arise in this case, because the child had been ill for seven weeks when I first saw it. Had I seen it at the beginning of the illness of course I should have given an injection. Congenital laryngeal stridor occurs earlier in life than the age of this child, and for this reason I did not refer to it in discussing the case. In

regard to Dr. Koplik's remark, I said that the dyspnea had been so intense prior to the intubation that the costal cartilages had been distorted and remained bent for twenty-four hours after the dyspnea was relieved. I did not mean that the dyspnea continued. We believe that we have a tetany which is continuous, and another form which is intermittent. If laryngismus stridulus is, as some believe, a form of tetany, we can easily understand that a continuous form of it could occur. From this broader point of view one might call the condition in the case I have reported a persistent laryngismus stridulus.

A NOTE ON THE TREATMENT OF GASTROINTESTINAL HEMORRHAGE IN THE NEWLY-BORN BY SUPRARENAL EXTRACT.

BY L. EMMETT HOLT, M.D.,
New York.

On February 26, 1901, I was asked by Dr. J. H. Moore, of New York, to see with him a baby twenty-four hours old, who was reported to be vomiting blood and also passing it from the bowels. I found a large, well-developed infant, weighing nine pounds at the time of birth. The labor was reported to be a natural one. Up to the time the infant was sixteen hours old it appeared in every respect quite normal. It then began to vomit blood, and this was repeated at intervals of a few minutes until I saw the child, about eight hours after the beginning of the symptoms.

The patient was the fifth child; the first was still-born; the second and third were alive and well; and the fourth died from hemorrhage, which began the second day after birth and continued up to its death on the fourth day. There was no suspicion of syphilis in either of the parents, both of whom were strong, healthy-looking people.

On examination there were found fine petechial spots about the neck and upper part of the chest, and one larger hemorrhagic spot over the left shoulder; many minute punctate hemorrhages were seen on the mucous membrane of the mouth. The amount of blood vomited each time was from a few drops to a teaspoonful. Most of it was of a dark-brown coffee-color mixed with mucus. Three or four large towels were shown which were pretty well covered with this material. There was no bleeding from the nose and no sign of a bleeding surface in the mouth or pharynx. The bowels had moved three or four times, the discharge being a little darker than normal meconium. The general condition of the baby was excellent. It was strong and vigorous, the heart and lungs were normal and no bleeding was

taking place from the cord. Several times during my stay in the house, I had an opportunity to see for myself the vomiting of blood. There seemed every reason for believing that the hemorrhage came from the stomach and it was decided to try the effect of suprarenal extract. One grain of the saccharated extract was ordered every hour, suspended in water.

This was given regularly for six or eight hours, after which the dose was reduced by Dr. Moore to half the quantity, on account of the difficulty in swallowing, owing to the astringent effect opon the pharynx and esophagus, and the intervals were made longer.

Twelve hours after beginning the suprarenal, the hemorrhages entirely ceased and did not recur. The child then began to take its food normally, and when forty-eight hours old was nursing as well as any child. By the fifth day the minute hemorrhages beneath the skin had disappeared as also those on the mucous membrane of the mouth. In all the child took twelve grains of the suprarenal, and of this ten grains were retained. The entire period of the hemorrhages lasted about thirty hours. The subsequent history of the child was uneventful. The cord separated in the usual time and no bleeding occurred. A report received three months later stated that the child was well in every respect.

No one realizes more than I do how uncertain are deductions drawn from a single observation, and my object in reporting this case is chiefly to call attention to the use of suprarenal extract in this condition, with the hope that a larger trial may be given to it in order to determine of how much value it really is. In my own experience, few things have been more unsatisfactory than the treatment of severe gastrointestinal hemorrhages in the newly-born. Occasionally a case recovers without active treatment, but the large majority go from bad to worse, and even children previously robust usually succumb after two or three days. In the case reported there seemed no escape from the conclusion that the drug given as above really benefited the child.

I have not had an opportunity to try it a second time, but Dr. Moore has reported to me a case of another child in which it was used. This was briefly as follows: The child began bleeding at two days. The suprarenal was used as in the other case reported, one grain every hour. The gastrointestinal

hemorrhage was controlled, but there was extreme bleeding beneath the skin and possibly a meningeal hemorrhage also, as the fontanel became very much bulged and the baby developed convulsions and died. In such a condition a fatal result was inevitable under any treatment.

The solution of the active principle, adrenalin, is possibly to be preferred for internal use to the powder, as one grain of the extract makes a rather large dose for a young infant. Whether any marked effect upon general hemorrhages is to be expected by internal administration of the extract is problematical, but its local effect seems to be proved in the case reported.

DISCUSSION.

DR. MORSE.—I have recently seen a case of hemorrhage in the new-born in which I used suprarenal extract locally. The baby was born, after a difficult forceps operation, and was wounded on both cheeks. On the following day hemorrhage occurred from one of these wounds, and later there was hemorrhage into the orbit and the child vomited some blood. On the second day the dry powdered extract was applied to the oozing surfaces with apparently no results whatever, and it became necessary eventually to use Monsell's solution and digital pressure. The child recovered perfectly. It seemed to me a favorable case for the use of the extract, but it failed completely.

DR. ROTCH.—The use of suprarenal extract, if it proves to be valuable, will be an important addition to our methods of treating hemorrhage in the new-born. These cases are often not hemophilia but hemorrhages possibly due to infection in which there is more or less of a tendency to get well without the aid of any drugs. The cases of hemophilia almost invariably die. There should, however, be a sharp distinction between the constitutional and supposed hereditary bleeding from hemophilia and those cases of hemorrhage occurring at birth and apparently of a local and infectious class, the hemorrhagic disease of the new-born.

DR. WILSON.—It seems to me it is most important in estimating the value of a therapeutic measure that as clear a conception as possible should be had of the natural history of the disease under consideration. As Dr. Rotch has pointed out, there are two essential conditions which are quite different in the origin of these hemorrhages, and in some there is a tendency to spontaneous recovery. In a case of this kind it seems to me

an improper estimate of the value of a therapeutic agent might arise.

DR. HOLT.—I would like to say a word about the use of the term hemophilia. It seems to me that is a misnomer when applied to the hemorrhages in the new-born. This disease, it is well known, has no relation to the hereditary disease, hemophilia, and the same term should not be applied to them. The hemorrhagic disease that occurs in the new-born is a self-limited one, probably of infectious origin, and the children either die or recover completely.

DR. CHAPIN.—Do I understand the last speaker to say that hemophilia does not occur in the first two or three weeks of life?

DR. HOLT.—It rarely appears until after the first year.

DR. CHAPIN.—In a case I saw there was the family history, and the child had hemorrhages shortly after birth.

I am very glad to know of the possibilities of good from the use of suprarenal extract and I shall certainly try it.

DR. CARR.—In a recent communication from Dr. Bates, whose experimental work on suprarenal extract has been so valuable, the statement was made that the great fault in the use of the extract has been in not using the aqueous solution. To get the best result he states that we should dissolve the powder, and he thinks its effect will be noticed within ten minutes in every case. That may be a little enthusiastic, but there is no doubt that when the hemorrhage can be reached locally the effect of the suprarenal extract is most satisfactory.

PYLORIC STENOSIS IN INFANTS, WITH A REPORT OF CASES.

BY E. W. SAUNDERS, M.D.,

St. Louis, Mo.

Judging from the increasing number of cases of hypertrophic pyloric stenosis published annually, the conclusion is warranted that this anomaly is by no means rare. The literature of this subject is quite extensive, and has been carefully reviewed by Meltzer, Pritchard, Abel, Nicoll and others.

The clinical phenomena are fairly uniform and typical, and the diagnosis offers little difficulty in the later stages.

After a certain interval from birth, varying from a day to three months, the infant, without apparent cause, begins to vomit. This is usually attributed to simple indigestion, or to some fault in the mother's milk. Under careful regulation of the diet and the administration of antemetics, improvement, if it occurs at all, is only temporary. The vomiting becomes worse and is projectile. Large quantities of milk are thus ejected, more than can be accounted for by the previous feeding. Obstinate constipation ensues. The infant more or less rapidly loses in weight.

The physical signs may be entirely negative in the beginning. A palpable tumor at the pylorus is exceptionally found. Progressive dilatation of the stomach occurs. On inspection of the abdomen the upper zone will be observed to be bulging, and contrasts strikingly with the depressed lower zone. The bulging of the epigastrium subsides visibly after a paroxysm of vomiting. Peristaltic waves are visible over the epigastrium after the abdominal walls become attenuated.

Examination of the gastric contents reveals the fact that the stomach does not empty itself in one or two hours. In some cases hydrochloric acid is in excess, but more often it is diminished or almost absent.

Coincidently with the gastric dilatation, the signs of gastric catarrh may become manifest. The contents of the stomach

show evidences of decomposition; the organic acids, lactic and butyric, being present. Large quantities of mucus are vomited, perhaps once in twenty-four hours, or once in two days. The condition of the infant grows steadily worse, the distress is more severe and the fatal issue is inevitable.

Such, in brief, is a clinical picture of this disease in its worst form. Post-mortem the pylorus is found to be very much thickened. The pyloric lumen is stenosed to a varying degree. The hypertrophy at the pylorus is composed principally of circular muscular fibres; but the longitudinal layer, the mucous and submucous coat also partake of this overgrowth. The accompanying table is taken from Batten's article and shows the relative enlargement. Cases I. and III. are normal at the pylorus. Numbers II. and IV. are hypertrophic.

Case	Age	Method of Preparing	Thickness of whole wall	Whole muscle laye	Circular muscle layer	Long muscle layer	Sub-mucous	Mucous	Lumen of Pylorus
1	16 Weeks	Müller fl. and freezing	1.5 mm.	1.05 mm.	0.75	0 3	0.2	0 25	
2	14 Weeks	do do	4.7	3.4	2.6	0.8	0.9	0.4	
		Necropsy	5.25	4.5					3.5 mm.
3	12 mos	Müller fl. and freezing	2.31	1.55	1.1	0.45	0.26	0.5	
4	11 mos	Formol and Celloid'n	4.2	2.9	2.5	0.4	0.5	0.8	
		Necropsy	5.5	4.	3.	1.			4 mm.

The pyloric opening varies in size, but the diameter of the canal at the necropsy is hardly a fair measure of its possible size during life. The muscular wall of the stomach is also very much thickened.

As illustrating the disease in all its severity I herewith give the report of a case which I saw in consultation. The history was kindly furnished by the physician in charge, Dr. W. B. Moore. I also take the liberty of demonstrating the stomach removed at autopsy.

CASE I. "The subject of this report is a female child born of healthy parents July 4, 1900. The labor was three weeks prema-

ture, but otherwise normal. Five days after birth the child had frequent green stools, one stool containing mucus and a slight amount of blood. There was some tenesmus but no vomiting. The nurse gave a small dose of calomel followed by castor oil, and later one grain of bismuth subnitrate every few hours. A physician was not summoned. Thereafter the baby did well until July 22nd, eighteen days after birth, when it became restless, cried frequently and vomited after nursing. I saw it for the first time the following afternoon. It was small but fairly well nourished and was crying as if in pain. The skin was slightly icteric and the fontanelle was depressed. Inspection of the mouth and fauces showed nothing abnormal. Nothing was found on examining the chest and abdomen. The temperature was normal. The nursing habits were good with the exception that the child was allowed to remain too long at the breast. The bowels had been evacuated several times, but the urine had not been passed for fifteen hours. The last urine voided showed a quantity of brick-dust deposits on the napkin. The mother was told to shorten the time of nursing. A diuretic was prescribed. The next day the child was quieter, although it was still restless and cried frequently. During the night it had vomited but once, but the mother thought the amount surprisingly large. Two small stools were passed. The passage of urine had been free but the brick-dust stains were still present. On the third day of the attack the child vomited after each nursing. The vomiting did not occur immediately, but was from one to two hours after the nursing. The vomited matter was free from curds and contained mucus. The quantity was such as to attract the mother's attention, who spoke of it as a 'double handful.' The child seemed quite sick before vomiting, but afterwards became brighter and was again eager for the breast. Failing nutrition began to manifest itself. During the day several small, dark green stools were passed, which seemed to contain only mucus and bile. The temperature was normal. Potassium bromid gr. v, was given by the rectum, and was repeated in four hours. The breast milk was temporarily withdrawn and a sample taken for examination. On the fourth day Dr. John Zahorsky saw the case with me. The nurse reported that sometime after giving the potassium bromid by rectum a normal yellow stool had been passed. The vomiting had continued unchanged. On examining the abdomen Dr. Zahorsky

discovered a tumor in the epigastric region, which was hard and readily escaped from under the fingers. It gave the impression of an intussusception of the transverse colon, but this was excluded by the absence of the characteristic stools and tenesmus. Dr. Carl Fisch reported that the milk contained 4 per cent. proteids. Minute doses of creosote were ordered. Normal salt solution was given by the rectum. In order to dilute the breast milk the mother was ordered to give the child albumen water just before allowing it to nurse. The fifth day the vomiting continued the same; the emaciation was increasing. The tumor which was felt on the preceding day could not be found. One small stool of mucus and bile was passed. The temperature was 99.5°. In order to exclude any infective process a blood smear was examined. Beyond a slight lymphocytosis it was normal. In order to determine the effect of a cathartic, gray powder was administered. After it was given the vomit was bile stained. In consequence Dr. Zahorsky and I concluded that there was an obstruction, from some congenital defect, but owing to the presence of bile in the vomit, that it was located below the pylorus. On the sixth day the symptoms continued unchanged and the emaciation was increased. Dr. E. W. Saunders was asked to see the child and, after considering the symptoms, made a diagnosis of pyloric stenosis, in which Dr. Zahorsky and I concurred. One grain of chloral was ordered to be given by the rectum twice daily. A grave prognosis was made. The parents refused operation, which was proposed, and were unwilling that the stomach tube should be used. On the seventh day it was noticed that the stomach was easily palpable. The lower border was made out by palpation and percussion at the level of the navel. During the day the child retained two nursings taken two hours apart, and vomited the whole three hours after the last. On the eighth day the lower border of the stomach was found 1 cm. below the navel. The ninth day slight vomiting occurred after each nursing, but in the afternoon the mother reported that enough sour curdled milk was vomited to wet two diapers thoroughly. From the tenth to the fifteenth day the child grew more and more emaciated. The vomiting occurred at longer intervals but in larger quantities. Each day one or two very small brown stools were passed. The temperature ranged from 99° to 102.5°. On the fifteenth day the stomach was plainly visible. Peristaltic waves

passed over it at intervals of two or three minutes. There was apparently an hour glass contraction, the stomach bulging on either side, with a sulcus in the middle. The child grew worse until the eighteenth day, when it died in an extremely emaciated condition.

"Permission was obtained to open the abdomen and remove the stomach. The peritoneum, liver and spleen were apparently normal. The intestines were empty. The pylorus immediately attracted attention owing to its almost cartilaginous consistency. The stomach contained an ounce of milky fluid. The mucous membrane was slightly congested, and thrown into longitudinal folds. The muscular coat of the stomach was hypertrophic, averaging 2 mm. in thickness. The hypertrophy was greater at the antrum pylori than elsewhere. A probe 2 mm. in diameter was passed through the pylorus, which began as an abrupt narrowing of the antrum pylori and terminated as abruptly in the comparatively wide duodenum. The length of the pylorus was 2.5 cm. and its muscular layer was 6 mm. in thickness. The mucous membrane of the pylorus was thrown into longitudinal folds. An histologic examination was made by Dr. Fisch, who reported that the hypertrophy was confined to the circular muscular layer of the stomach and pylorus. The other tissues were normal.

"The misleading symptom in this case was the presence of bile in the vomitus. It is evident that the violent contraction of the abdominal muscles during emesis may force a small quantity of fluid intestinal contents into the stomach."

But all cases do not run such a rapid course. The fatal issue may be warded off and the infant may recover. It is my purpose to draw particular attention to these milder cases and discuss their successful management.

CASE II.—Baby. Was nursed at the breast for two months. Vomiting commenced when the infant was six weeks old. This hyperemesis gradually became more aggravated. The infant was taken from the breast by the physician then in charge and placed on artificial food. Various modifications of cow's milk and patent foods were used but with little success. At times an improvement would be noticed for a short time, then a relapse would occur. When about five months old it first came under my care. Found an emaciated infant weighing about nine pounds. No pulmonary, cardiac, or nervous abnormalities.

The stomach was enlarged, reaching below the umbilicus. Vomiting would occur after each feeding. Usually small quantities would be successively vomited until most of the food ingested would be lost. No tumor was palpable at the pylorus. Free hydrochloric acid was not demonstrable in the vomitus by Guinzburg's test. Considerable mucus was present in the vomit at times.

The little patient was put on a whey and cream mixture. It improved for a few days, and then relapsed. Inquiry elicited the fact that the mother was giving 8 to 9 ozs. of milk at each feeding. The stomach was washed out once only, and the quantity of food restricted to 3 ozs. The infant then began to gain in weight and the vomiting gradually ceased. The infant is one and a half years old now and a very well developed child.

The recovery of this patient dates from the time it was put on a whey mixture. The coagula formed by the casein seemed to choke up the pylorus. No medicinal agent was employed except a few doses of chloral at the onset.

CASE III.—Baby. Female. Weighed eight pounds at birth. She was breast-fed for three weeks, but did not thrive. A severe mastitis developed in the mother and the attending physician ordered the infant to be taken from the breast and placed on condensed milk. Two days later the patient commenced to vomit. This persisted in spite of treatment. The physician ordered modified milk, Mellin's food and malted milk, successively, but the hyperemesis persisted. Throughout all this time the baby was obstinately constipated. It was then put on peptonized milk, and seemed to improve somewhat. A wet nurse was finally procured, but the vomiting became more violent. For one month this nurse tried to nourish the infant, but it rapidly lost weight until it weighed only five pounds at the age of three months. The physician in charge then discontinued feeding by the stomach, and rectal alimentation only was employed for two weeks. The infant, after this prolonged abstinence from stomach feeding, was able to retain a little peptonized milk. She was five months old when she came under my care, and weighed five pounds. The diet was peptonized milk, ½ oz. at a feeding, 6 ozs. per diem. I increased the allowance to 1 oz. at a feeding, 18 ozs. per diem. If she took more than

1¼ ozs., she invariably vomited the whole feeding. The thickened wall of the stomach could be plainly made out through the thin wall of the abdomen. I could not be certain that it was a case of pyloric stenosis at that time, because of the absence of dilatation, the symptoms having persisted so long.

The vomiting almost ceased and the infant gained in weight. A wet nurse was procured after some weeks, but her milk caused a relapse. At times, from very slight causes, stagnation of the gastric contents took place, followed by hyperesthesia of the gastric mucous membrane and pyloric spasm, and the condition of the little sufferer grew worse.

In April a persistent attack of vomiting began. Operation was finally advised, but as the infant improved before she was taken to hospital, it was postponed.

May, 1901, the infant is fourteen months old and weighs ten pounds. She is taking a mixture of 8 per cent. cream with a dextrinized food, gaining in weight, does not vomit, and looks very well. The stomach, when moderately distended, reaches ½ cm. below the umbilicus.

November, 1901.—At twenty months the weight is fifteen pounds, and the child, although a midget, is well-nourished, and has had no relapse for some months. The typical pear-shaped abdomen has taken on the normal appearance of infancy, the flanks and hypogastrium are well rounded and resonant.

The diagnosis in this case was for a long time in doubt, but the gastric hypertrophy, first observed, followed by a gradual dilatation, the peristaltic waves seen only after the case had been long under observation, the typical shape of the abdomen, placed it finally beyond a doubt.

The puzzling features were the intermittency of the vomiting, and the presence of true diarrhea occasionally at the onset of a relapse.

It seems reasonable that we should recognize a class of cases of pyloric difficulty more or less intermittent in its symptoms, which is clearly not congenital.

In adults we see a prolonged series of attacks of so-called gastralgia, which may be fairly attributed to an irritated pylorus which is thrown into spasm whenever an unwonted chemical or mechanical stimulus is applied, by reason of injudicious diet or indigestion.

This case illustrates the fact that pyloric spasm is a great factor in the production of emesis. Fermentation of the gastric contents with the resultant irritation of the pyloric mucous membrane, increases the spasm.

It is not rare that infants come under observation, who regurgitate so much of their food daily, that their growth is seriously impaired. Possibly many such patients suffer from a thickened pylorus.

Pfaundler holds that pyloric spasm is the principal cause of the symptoms in these cases. Thomson has advocated the theory that hypertrophy is always secondary to spasm. In these milder cases the spasmodic element certainly seems to be the principal factor.

Very interesting are those cases of persistent vomiting associated with hyperchlorhydria. Knopfelmacher has recently reported such a case. In his case the symptomatology was similar to that of pyloric obstruction. Gastric motor insufficiency was also demonstrated. But the diagnostic phenomenon was the presence of an excess of hydrochloric acid in the gastric juice. The infant recovered on whole milk, the casein of which neutralized the excessive acid.

A similar case occurred in the Bethesda Foundling Home, a brief history of which is as follows:

CASE IV.—Fannie. She was a healthy infant at birth. When several weeks old she began to vomit after every feeding. She was fed throughout on laboratory milk. At first she was put on a mixture containing proteids, 1; fat, 2; sugar 6 per cent. When about three months old the food was changed to proteids, 1.75; fat, 3.50; sugar, 7. The vomiting persisted for several months in spite of remedies and dietary changes. No enlargement of the stomach was demonstrated. The digested milk vomited invariably gave a very strong reaction of hydrochloric acid by Töpfer's test. A great excess of this acid was always present after nursing. The gastric contents were so irritating as to produce a severe dermatitis on the neck, which resisted all treatment until the vomiting ceased. The nutrition suffered for a long time. When the infant was nine months old she was placed on undiluted cow's milk and the vomiting almost completely ceased. She is now a healthy baby.

As in the case reported by Knopfelmacher the vomiting ceased on the administration of undiluted cow's milk.

Sometimes in cases which do not improve on raw milk, vomiting ceases when it is heated to 212°. Where whole milk is not borne at all, white of egg will sometimes answer the purpose.

These cases, from the prognostic and therapeutic point of view, must be sharply differentiated. Whenever persistent vomiting occurs, the presence or absence of hydrochlorhydria must be established. Patients with pyloric stenosis should be fed by whey mixtures, or fully peptonized milk, while pyloric spasm from an excess of hydrochloric acid, must be treated by an excess of proteid to neutralize the acid. In the case of Baby L., at the age of five months the milk had to be peptonized for forty minutes. No wet nurse succeeded until one was found whose child was thirteen months old and whose milk showed 0.8 proteids.

Dr. Zahorsky furnished me with the history of the following case which illustrates what can be accomplished by careful dieting.

CASE V.—Baby F. Male. Age four months. Weighed about nine pounds at birth. The labor was normal. For the first two weeks the baby thrived. About the fifteenth or sixteenth day vomiting set in. This vomiting persisted and became daily more aggravated. The physician in charge prescribed various remedies with little effect. The milk was rejected from ten minutes to three hours after nursing. The bowels became obstinately constipated. The infant commenced to lose in weight, and at the end of four months weighed about eight pounds.

At varying intervals the vomiting would improve. The family physician put it on a variety of artificial foods and milk mixtures, but the vomiting grew worse. Finally he resorted to rectal feeding exclusively and under this method the infant seemed to do best.

The infant came under my care when four and one-half months old. Examination showed a poorly nourished child, weighing about seven pounds. The face was thin and the skin hung in folds; slept very well, cried little, and passed a considerable quantity of urine. No food had been given by the stomach for twelve hours previous, and during this time no vomiting had occurred. The parents had not noticed any bile in the vomitus at any time. Examination of the mouth, lungs, heart, liver and spleen revealed nothing abnormal. Temperature of the body

was 98°. The abdomen was flat, with slight bulging of the lower half, but as a nutrient clyster had been given a short time previously the swelling was attributed to this. When the lower bowel was emptied the swelling subsided. No tumor was palpable in the epigastrium, and no enlargement of the stomach was demonstrable, but later, when the stomach was distended, it was found that the greater curvature extended to the umbilicus. Moreover, it was observed that the upper half of the abdomen was relatively very prominent when the colon was empty. The diagnosis of hypertrophic pyloric stenosis seemed most reasonable.

The infant was given whey, which it took greedily. A little cream was added to this in one or two days, but the addition of cream caused distress, crying and vomiting. The vomitus had the odor of butyric acid. No excess of hydrochloric acid was present. The treatment consisted of rigidly maintaining the whey mixture, which could not form a coagulum in the stomach. As an antispasmodic atropin was given in doses of 1-1000th of a grain three times a day. This was given for the purpose of preventing pyloric spasm. The food given was pure whey, to which a little sugar was added. To make up the deficiency in fat, 15 drops of cod-liver oil were given after each meal.

The infant improved and commenced to gain in weight. In order to increase the strength of the food peptonized milk was mixed with the whey. The peptonized milk was gradually increased until the infant took equal parts of whey and peptonized milk representing a mixture of 2½ proteids, 2 fat, and 7 per cent. sugar. The quantity given was at first 2 ozs., which was gradually increased to 4 ozs. Now, two months after beginning the treatment, the baby weighs twelve pounds, regurgitates very little food, seems happy, looks well, has normal passages and continues to thrive. About three weeks ago it passed through an attack of influenza with high fever, but it regained its weight rapidly when the fever subsided. Lavage of the stomach was not believed to be necessary in this case after the long absence of food from the stomach. The atropin was continued only for about two weeks.

PATHOGENESIS.—This is unknown. Cautley believes that the condition is due to a congenital redundancy, or a prenatal overgrowth of muscular tissue.

Thomson believes that the trouble is a functional disturbance of the nervous system leading to a spasm and secondary hypertrophy of the pylorus. Pfaundler claims that the condition is primarily a spasm, and the hypertrophy is overestimated on account of the extreme contraction. As has been said, "It seems reasonable to combine these views so far as to believe that there is some congenital hyperplasia of the pyloric sphincter and that spasm supervenes upon this, and is largely responsible for the symptoms manifested.

TREATMENT.—Medicinal and dietetic measures should, in all cases, be first employed. The indications are as follows:

First.—The administration of some medicinal agent which shall overcome to a greater or less extent the violent contractions of the pylorus. Among the drugs to be recommended are belladonna, bromids and chloral. In case no. 1 chloral had only a temporary effect; atropin seemed very successful in case no. 1. Opiates should not be given, as the motor function of the stomach is thereby impaired.

Second.—The treatment of the secondary gastric irritation. This results from the stagnation of food and should be treated by washing out the stomach, and by giving the stomach rest; rectal feeding should, therefore, be resorted to from time to time and for twenty-four or more hours, nothing but water given by the stomach. When food by the mouth is again allowed, the stomach should be washed out occasionally to remove a possible residuum of undigested food.

Third.—The diet of the child should consist of food which forms no coagulum in the stomach. This point has not been sufficiently insisted upon. Milk or any food containing undigested casein will not answer, consequently the mother's milk is usually unsuitable, while the milk of a wet nurse in advanced lactation will succeed. Whey or peptonized milk or a mixture of both is generally the best food. The deficiency in fat should be supplied by cod-liver oil. A very small percentage of cream can be gradually added. If the coagulum formed by the cream causes distress or increases the vomiting, it must be completely predigested. A mixture of whey and predigested milk perfectly agreed with the patient last named. It is well to aid the motor power of the stomach by gravity, hence, after nursing the infant should be placed on its right side. If the diet is per-

fectly fluid some nourishment will pass through the pyloric opening.

The end to be accomplished is hypertrophy of the gastric wall without dilatation, hence the quantity of food should not be large. Gaseous distension of the stomach should by all means be prevented. I cannot agree with Meltzer, who advocates giving large quantities of milk in the first stage. This must inevitably lead to ectasia. A stomach is not stronger when over distended.

When it is seen that the infant is failing in spite of rational treatment, surgical intervention must be advised.

As to the method of operation, we must leave the decision entirely with the surgeons, until sufficient material is furnished on which we can base some conclusions.

Schmidt has added a third case to the two successful operations recorded by Nicoll and Abel respectively. He performed a digital dilatation, but whether he opened the stomach, or simply invaginated the wall, as advised by Hahn, the reporter does not say.

1635 S. GRAND AVENUE.

DISCUSSION.

DR. HOLT.—I think one needs to be very careful in drawing any conclusions from the finding of a contracted stomach. I have seen a great many times at autopsy a stomach as contracted as this one, where I am absolutely sure there was nothing the matter except that the stomach was empty. I have found these stomachs in children all the way from a few weeks to several years of age. I think we should make a mistake in calling such a condition a result of hypertrophic pyloric stenosis. I am getting somewhat skeptical about this condition, because so many cases have been reported lately. The hypertrophy at the pyloric orifice in the specimen shown is certainly very marked, but I think we should not attach any importance to the apparent thickening and prominent corrugations of the stomach wall.

DIPHTHERIA OF THE CONJUNCTIVA TREATED BY ANTITOXIN.

BY L. EMMETT HOLT, M.D.,
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Cases of conjunctival diphtheria treated by antitoxin are not so numerous but that the report of an additional one may possess some interest.

The patient, an infant six months old, was admitted to the New York Foundling Hospital, May 18, 1901, without any history. Examination showed swelling and redness of the right eye, especially of the upper lid, to such a degree that the child was unable to open it. On everting the lid a thick yellowish gray patch of pseudo-membrane was seen upon the conjunctiva, covering about one-half of the lid. This membrane was very adherent. There was evidence of considerable conjunctivitis and the cornea was slightly cloudy. There was no membrane in the throat or elsewhere, and the general symptoms were those of moderate prostration, temperature 102°.

A fresh smear and afterward a culture from the conjunctiva showed diphtheria bacilli in numbers, and 2,400 units of antitoxin were administered. Ice compresses were applied and a solution of atropin dropped into the eye. The progress of the case was rapid and uneventful. In twenty-four hours the temperature had fallen to 100 .4-5°, and in forty-eight hours to normal. Improvement in the eye began almost immediately and in twenty-four hours a very decided change was seen. On the third day the swelling had so far diminished that the child could open the eye and the membrane was nearly gone. It did not disappear entirely until the sixth day. Diphtheria bacilli were found by cultures as late as the seventh day, but on the tenth day the eye was entirely well.

No more convincing demonstration of the value of antitoxin can be found than in cases like this, which formerly under any treatment known almost invariably went on from bad to worse, ending in the destruction of the eye.

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